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EVALUATION OF A POSSIBLE CAUSAL RELATIONSHIP BETWEEN FALLOUT DEPOSITION OF STRONTIUM 90 AND INFANT AND FETAL MORTALITY TRENDS

Edythalena Tompkins, et al

Bureau of Radiological Health
Rockville, Maryland

October 1969

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Evaluation of a Possible Causal Relationship between Fallout Deposition of Strontium 90 and Infant and Fetal Mortality Trends

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Evaluation of a Possible Causal Relationship between Fallout Deposition of Strontium 90 and Infant and Fetal Mortality Trends

by

Edythalena Tompkins
and
Morton L. Brown
Division of Biological Effects

October 1969

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Consumer Protection and Environmental Health Service
Environmental Control Administration
Bureau of Radiological Health
Rockville, Maryland 20852

FOREWORD

The Bureau of Radiological Health conducts a national program to limit man's exposure to ungainful ionizing and nonionizing radiations. To this end, the Bureau (1) develops criteria and recommends standards for safe limits of radiation exposure, (2) develops methods and techniques for controlling radiation exposures, (3) plans and conducts research to determine health effects of radiation exposure, (4) provides technical assistance to agencies having radiological health programs, and (5) conducts an electronic product radiation control program to protect the public health and safety.

The Bureau publishes its findings monthly in Radiological Health Data and Reports, and in the Environmental Health Series, Public Health Service numbered publications, appropriate scientific journals, and from time to time in Division-sponsored technical reports.

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John C. Villforth
Director
Bureau of Radiological Health

PREFACE

As early as 1963, Dr. Ernest J. Sternglass expressed his belief that radiation due to deposited radionuclides from fallout has increased the burden of diseases, such as leukemia, and altered the trend of infant and fetal mortality in the United States. More recently, through public forums, published articles, and presentations at scientific meetings, Dr. Sternglass has given added emphasis to what he considers a highly significant and disturbing finding in changing patterns of infant and fetal mortality, which he has attributed primarily to levels of ^{90}Sr in the environment.

Because of the magnitude of the problems implied in these allegations, the Public Health Service conducted an evaluation of the reports written by Dr. Sternglass. This critique, based primarily on Dr. Sternglass' report given at the 9th Annual Hanford Biology Symposium on May 5-8, 1969, in Richland, Washington, has resulted in a failure by us to agree with the scientific approach used by Dr. Sternglass in evaluating the data he has utilized in his report.

The Department of Health, Education, and Welfare is, of course, extremely concerned with the changes in infant mortality trends with no immediately obvious factors to account for them. We cannot support Dr. Sternglass' conclusions and ascribe changing patterns in infant and fetal mortality to the cause and factors invoked by Dr. Sternglass -- certainly not with the conviction or certainty required by most epidemiologists and statisticians. It is clear from a review of all the data that certain gaps in the knowledge of how environmental levels of ^{90}Sr may affect the genetic material of individuals still exist and further studies in this direction are probably warranted. However, nothing at the moment would indicate that any large-scale efforts to remove ^{90}Sr from milk or to employ other measures of this sort are warranted.

The Public Health Service will continue to evaluate all possibilities such as those raised by Dr. Sternglass as part of its continuing program in radiological health. We solicit the interest and comments from all those concerned with these matters of radiological health.

William A. Mills

William A. Mills, Ph.D.
Director
Division of Biological Effects

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EVALUATION OF A POSSIBLE CAUSAL RELATIONSHIP BETWEEN FALLOUT DEPOSITION OF STRONTIUM 90 AND INFANT AND FETAL MORTALITY TRENDS

INTRODUCTION

In April 1969, an article entitled "Infant Mortality and Nuclear Tests" by Dr. Ernest J. Sternglass was published in the Bulletin of the Atomic Scientists. Since that time, Dr. Sternglass has presented similar papers at two scientific meetings, has had many interviews on television and with the press, and has published several articles in journals and magazines. In each of these presentations and articles, Dr. Sternglass states that his research has proven a causal relationship between the deposition of ^{90}Sr from fallout due to atomic weapons testing and the decrease in the rate of downward trend of infant and fetal mortality. He also states that the slower rate of decline was caused by "excess" deaths that resulted from genetic damage due to incorporation of ^{90}Sr into the genetic material of the parents before the child was conceived.

The possible validity of a demonstrated genetic effect in man due to a low accumulated gonad dose resulting from fallout is of great interest to the Bureau of Radiological Health; consequently, a thorough review of the evidence presented by Dr. Sternglass has been undertaken. As the data published consist almost exclusively of graphic presentations, the evaluation has been, of necessity, limited to analyses of these charts and graphs. The paper which presents the most complete set of data and which includes all of the charts and graphs appearing in other articles is "Evidence for Low-level Radiation Effects on the Human Embryo and Fetus," printed in the Proceedings of the Hanford Symposium on "The Radiation Biology of the Fetal and Juvenile Mammal," May 5-8, 1969. A preprint copy of this article was used for this evaluation.

The data presented as evidence for a causal relationship between fetal and infant deaths and ^{90}Sr is divided into three main subheadings: Childhood Leukemia, Fetal Mortality, and Infant Mortality.

CHILDHOOD LEUKEMIA

The childhood leukemia data consist of presentations of various distributions of 55 deaths from leukemia in children 0 to 15 years of age (47 cases between 0 and 10 years) which occurred in the Albany-Troy, N. Y. area during the years 1952-1962. Dr. Sternglass has reported on these same cases in earlier papers in an effort to show a causal relationship between an "increased incidence" of childhood leukemia in the Albany-Troy-Schenectady area and the rainout of radioactive debris experienced in this area in April 1953. As in the earlier reports, Dr. Sternglass has not related the observed cases to the population at risk. Without these data it is not possible to determine whether an increase in incidence did indeed occur (Figure 1 - from Sternglass). Dr. Sternglass indicates that the population in the three New York counties, Albany, Rensselaer, and Schenectady, increased by only 10.5% in the years 1950 to 1960, and therefore could not account for the increased number of cases observed. However, the population at risk, that is, children between 0 and 15 years of age, increased by 40% in this metropolitan area during the same 10-year period.

Dr. Sternglass also makes the assumption that all the children who died of leukemia in Albany-Troy after 1953, and who were alive as of April 1953, were actually living in the fallout area and were, therefore, exposed to the fallout radiation. He then tries to demonstrate that the distribution of intervals between "exposure" and onset of leukemia in the Albany-Troy children is very similar to an analogous distribution for a group of children who died of leukemia and who had a history of therapeutic irradiation of the thymus gland (Figure 2 - from Sternglass). There are two major shortcomings with this comparison. For some reason, Dr. Sternglass omitted four cases of leukemia which developed in 1954 (within 1 year of the fallout) in the distribution of the Albany-Troy cases. The inclusion of these missing cases makes the distributions of time intervals for the two groups of cases quite different.

A more serious question is the validity of the comparison between the two groups of leukemia cases with regard to intervals between exposure and death. All of the children who had thymic irradiation were in early infancy at the time of exposure (usually less than 3 months of age). Thus, a distribution of time intervals for this group of children from exposure to leukemia is also a distribution of the approximate age at onset of the disease. The Albany-Troy cases, on the other hand, were between 0 and 10 years old at the time of the presumed exposure. Dr. Sternglass, therefore, compares an age-dependent distribution with an age-independent distribution and argues that the fact that they both peak at 5 years supports the thesis that the Albany-Troy cases were radiogenic.

Using the above argument, Dr. Sternglass continues by stating that there is a similar distribution of intervals in the children who were not yet conceived at the time of fallout and who later developed leukemia,

again supporting the radiogenesis of the disease (Figure 3 - from Sternglass). The assumption made here is that the parents of all of the children who were conceived following the fallout in Albany-Troy and who developed leukemia were residents in the area at the time of fallout and were thus exposed. By relating the time of onset to time of conception, Dr. Sternglass has again merely demonstrated the distribution by age of the leukemia cases. He could have noted that this distribution follows the known age distribution of the incidence of childhood leukemia, as he himself demonstrates in his next figure (Figure 4 - from Sternglass).

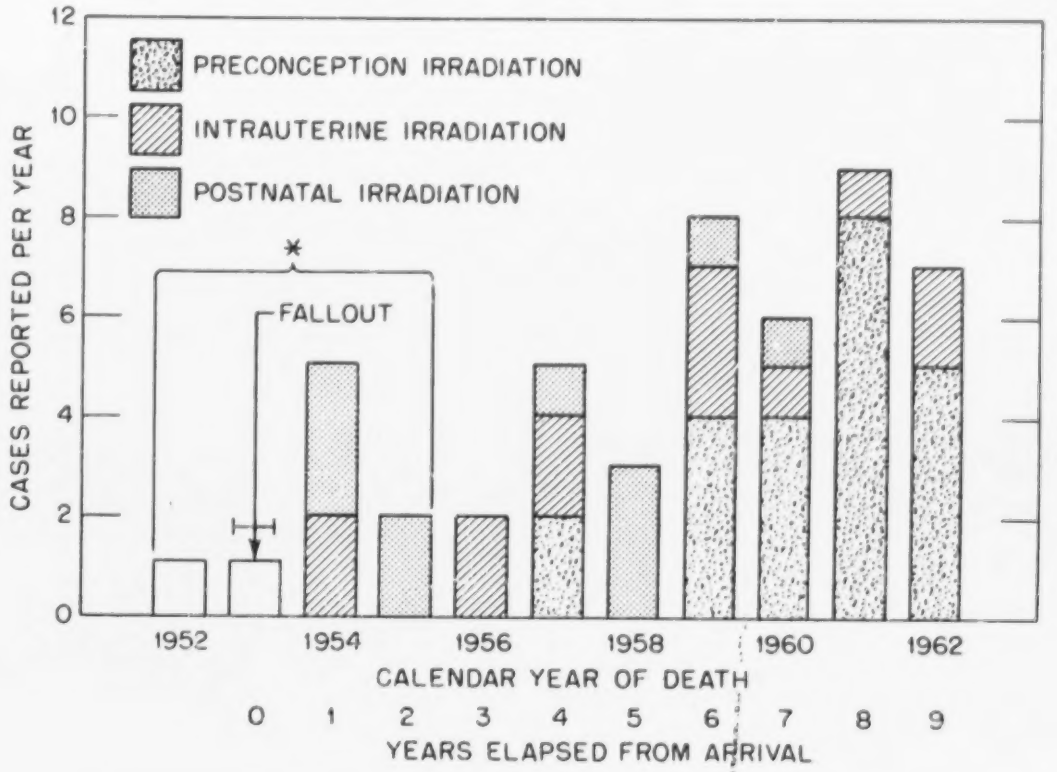


Figure 1. Number of leukemia cases reported per year in the Albany - Troy, N.Y., area for children 0 to 10 years of age from 1952 to 1962 for which complete data were given by Lade. Postnatal irradiation, children living at the time of fallout arrival in 1953; intrauterine irradiation, children in utero or early infancy in 1953 and 1954; postconception irradiation, children born after 1954. Since death generally occurred within 2 to 12 months after onset, the year of report or onset may be taken also as the year of death. The population in the three county area was 514,490 in 1950 and 568,407 in 1960, an increase of only 10.5%. (from Sternglass)

Dr. Sternglass states that the above data are "strongly suggestive of a causal relation between the increased incidence of leukemia and the radiation from the fallout in Albany-Troy," but goes on to say that even "stronger evidence" is found in a shift to an older age distribution of the cases reported post-fallout. He then proceeds to show a difference in the age distribution of cases reported in the 8 years before 1955, and the 8 years after 1954, by simply subtracting within age groups the number of cases observed in the earlier time period from that observed in the later time period (Figure 4 - from Sternglass). The result of such a calculation would be interpretable only if the relative number of children in each age group at each time period was constant. This is definitely not the situation in the population of children in the Troy-Albany-Schenectady area. The children between the ages of 0 and 10 in the period of 1954-1962 strongly reflect the well recognized post-war "baby boom" of the United States. For example, in this geographic area, between 1950 and 1960, the 1 to 2 year-old group increased only by 17%, while the 7 to 9 year group increased by 42% and the 10 to 13 year by 69%.

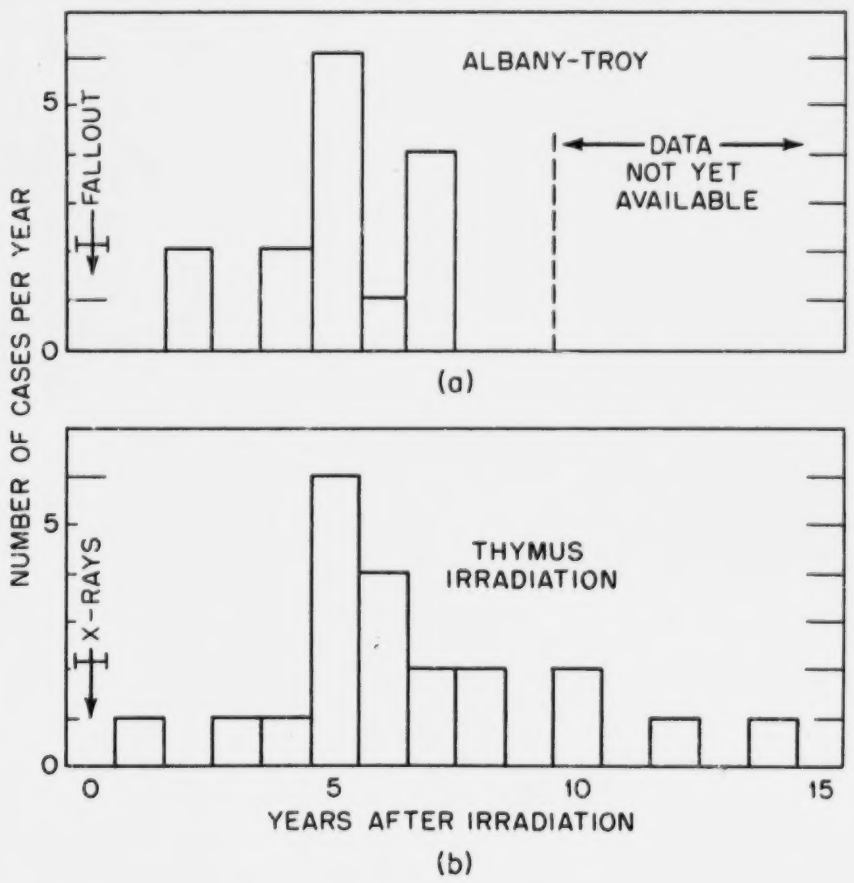
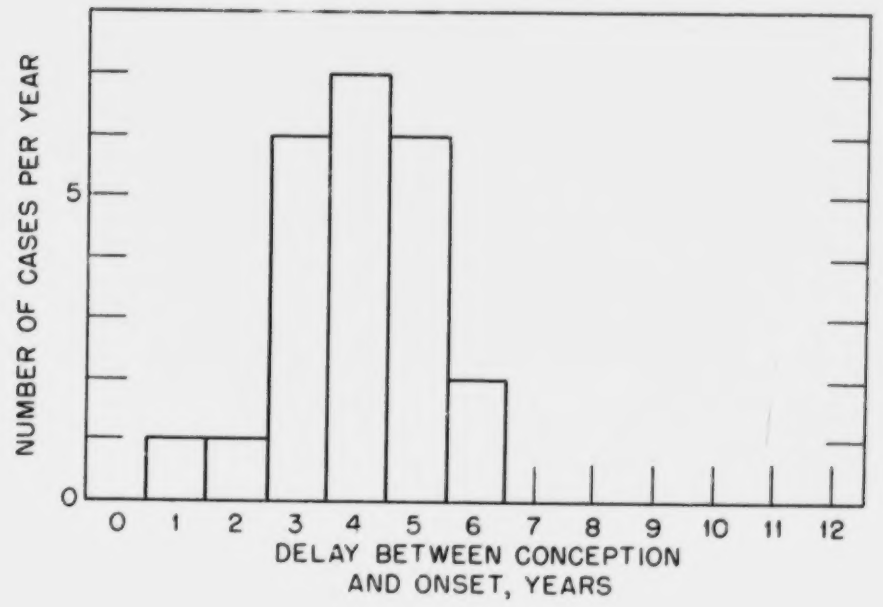
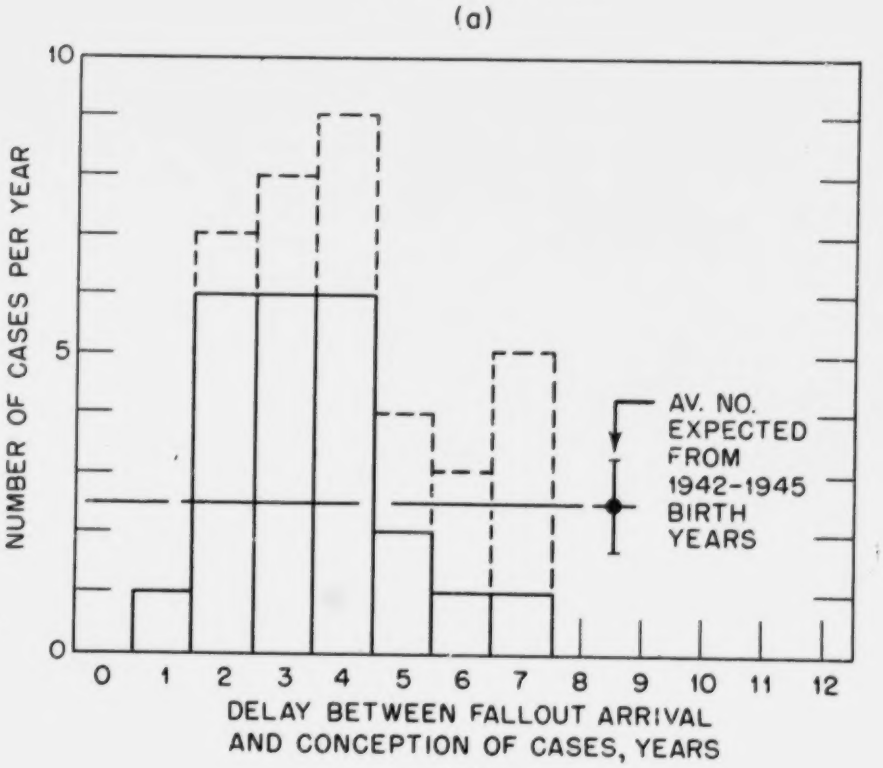


Figure 2. Patterns of onset of leukemia following irradiation. (a) Children 0 to 10 years old in Albany-Troy at the time of fallout arrival in 1953. (b) Infants who received X rays to their thymus. (from Sternglass)

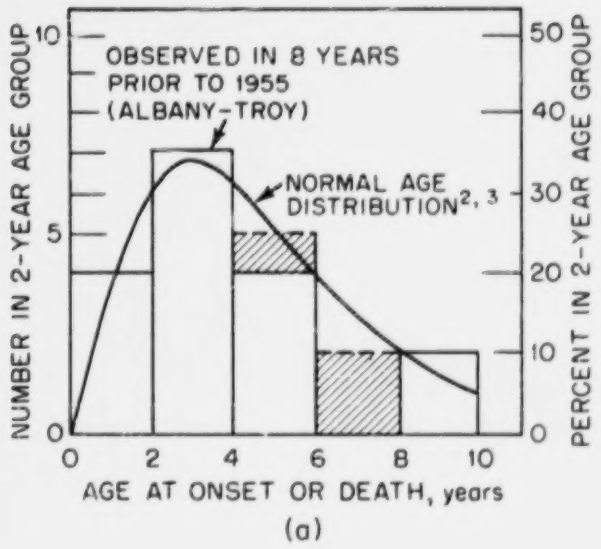


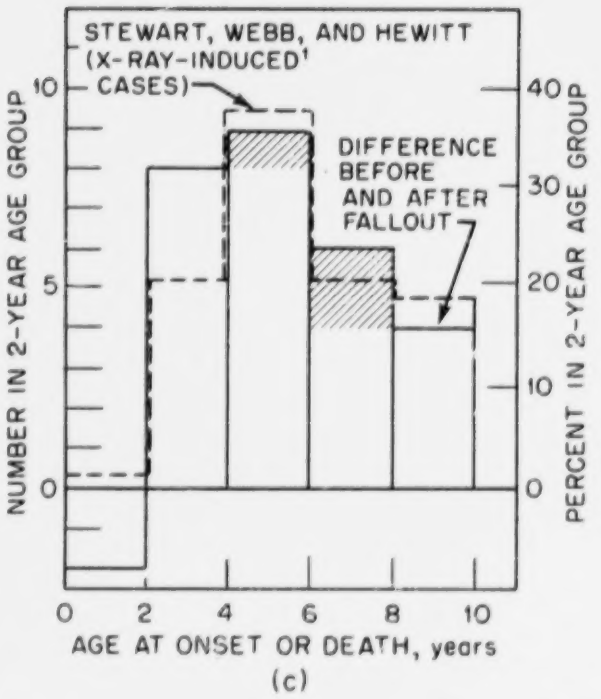
(a)



(b)

Figure 3. Cases of leukemia as a function of time after conception for cases born after 1954. (a) Delay between conception and onset. (b) Delay between fallout arrival and conception.


OBSERVED IN 8 YEARS PRIOR TO 1955 (ALBANY-TROY)
NORMAL AGE DISTRIBUTION[2, 3]
NUMBER IN 2-YEAR AGE GROUP
PERCENT IN 2-YEAR AGE GROUP
AGE AT ONSET OR DEATH, years
(a)


STEWART, WEBB, AND HEWITT (X-RAY-INDUCED[1] CASES)
DIFFERENCE BEFORE AND AFTER FALLOUT
NUMBER IN 2-YEAR AGE GROUP
PERCENT IN 2-YEAR AGE GROUP
AGE AT ONSET OR DEATH, years
(c)

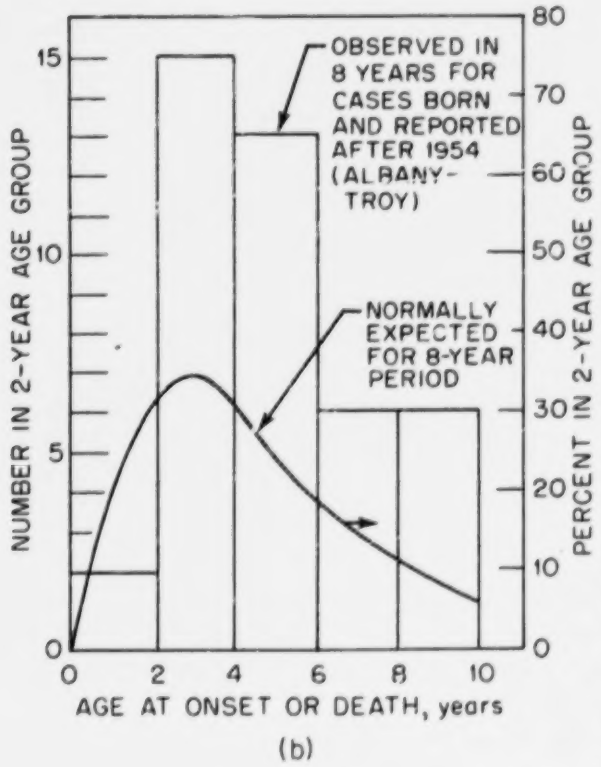

OBSERVED IN 8 YEARS FOR CASES BORN AND REPORTED AFTER 1954 (ALBANY-TROY)
NORMALLY EXPECTED FOR 8-YEAR PERIOD
NUMBER IN 2-YEAR AGE GROUP
PERCENT IN 2-YEAR AGE GROUP
AGE AT ONSET OR DEATH, years
(b)

Figure 4. Age distribution for leukemia cases in the 0- to 10-year age group for the Albany-Troy area. (a) Cases reported in an 8-year period before 1955. (b) Cases reported for an equal period after 1954. (c) Difference between (b) and (a) compared with the radiogenic age distribution for cases known to have been exposed to diagnostic X rays. Shaded areas in (a) and (c) represent a correction for three cases missing because no data for birth years prior to 1943 were available. For the two 8-year periods examined, the total number of births increased only 6%, from 93,030 to 98,797, compared with an increase of (22/20), or 110%, for the total number of leukemia cases. (from Sternglass)

The final evidence for an increase in leukemia due to the effects of fallout is a plot by years and selected age groups of all the cases of childhood leukemia reported for New York State for the years 1954 through 1966. Dr. Sternglass compares these cases with the observed average number of cases in the same age groups in New York State for the years 1950-1954 (Figure 5 - from Sternglass). Again, the fallacy of drawing conclusions based on a distribution of cases independent of the population from which they arise is evident. Sternglass observes an "excess" of cases and tries to demonstrate that these are primarily in the older age group, and that each rise can be correlated with a known weapons test some 5 years earlier. The population statistics for New York State for the census years 1950 and 1960 show an increase of 38% in the 0 to 14 year old children, with only a 7.5% increase in children under 1, but a 48% increase in the children 5 to 14. If one takes the approach which Dr. Sternglass follows, a 38% increase in the 0 to 14 year old group between 1950 and 1960 should lead to a similar percent increase in leukemia cases expected in 1960. As an estimate, one would expect 206 cases of leukemia in the same age group in 1960 assuming the same incidence of disease as in 1950. As 209 cases were the maximum observed in New York State in any of the years shown, this calculation makes it difficult to assume any increase in leukemia observed over that expected during the time period in question in New York State.

The dangers in postulating the existence of an association based on comparisons of number of cases observed over periods of time is well recognized by epidemiologists. Unless the cases can be related to a base population from which the cases were derived and comparisons then made of the observed rates, or unless the base population has been static, no trends of change over time can even be assumed. Even if such a trend could be demonstrated, an increase in an "exposed" population without a different pattern in "non-exposed" populations falls short of even demonstrating an association, let alone a causal relationship.

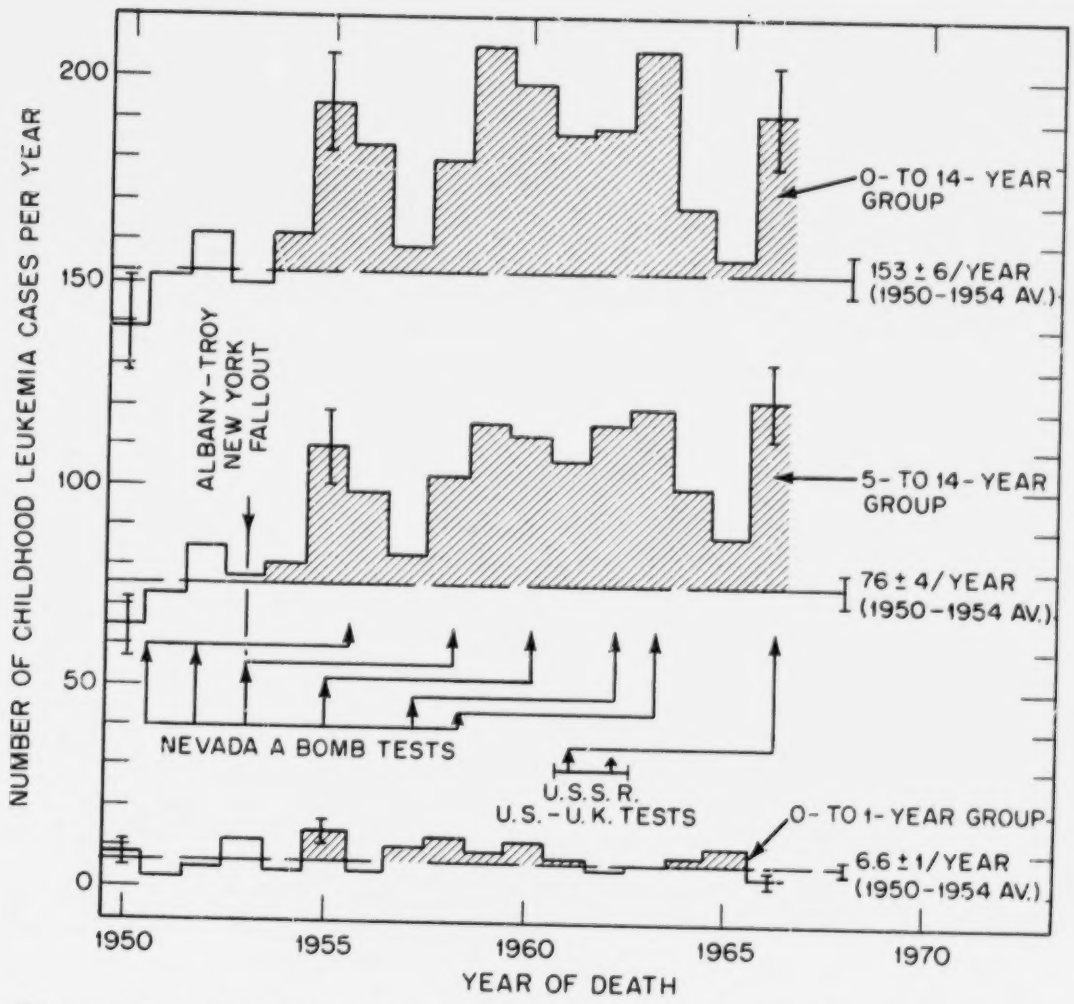


Figure 5. Variation in number and age distribution of childhood leukemia cases in New York State with time, correlated with atmospheric atomic bomb tests in Nevada and hydrogen bomb tests in the Pacific. Note that each rise in total number of cases 0 to 14 years old at death is almost entirely accounted for by the rise in the number of cases 5 to 14 years old, known to reflect radiation effects most strongly. Also, each such rise can be correlated with a known weapons tests some 5 years earlier, corresponding to the delay in onset observed in Albany-Troy and for children that received thymus irradiation. Vertical bars represent one standard error. (from Sternglass)

FETAL AND INFANT MORTALITY

The same basic analytic method is used by Dr. Sternglass in analyzing most of the data he presents to support his thesis that the changes in rates of decline of fetal mortality ratios and of infant mortality rates in the United States are associated with fallout from nuclear weapons testing. He assumes that a logarithmic transformation of the observed mortality rates for the years 1935-1950 is linear, and that a linear projection of this line would describe the trend in infant and fetal mortality over the next 16 to 17 years if there had been no deposition of fallout. He attributes all deviations from this projected line to the effects of fallout, particularly to the incorporation of ^{90}Sr into the genetic material of the parents. If this assumption is valid, it must follow that the patterns of fetal and infant mortality rates are independent of such recognized influences as epidemics, advancements in the medical sciences, improvement in prenatal care and nutrition, control of infectious diseases, and changes in socioeconomic conditions. The logarithmic transformation of the observed fetal mortality ratios and infant mortality rates for the United States have been plotted for all the years for which data are available (Figure 6). If one limits his attention only to the rates observed before the atomic age, it is apparent that there are many deviations from linearity between individual years as well as basic changes in trends of rates over time. Thus, this assumption appears to be untenable.

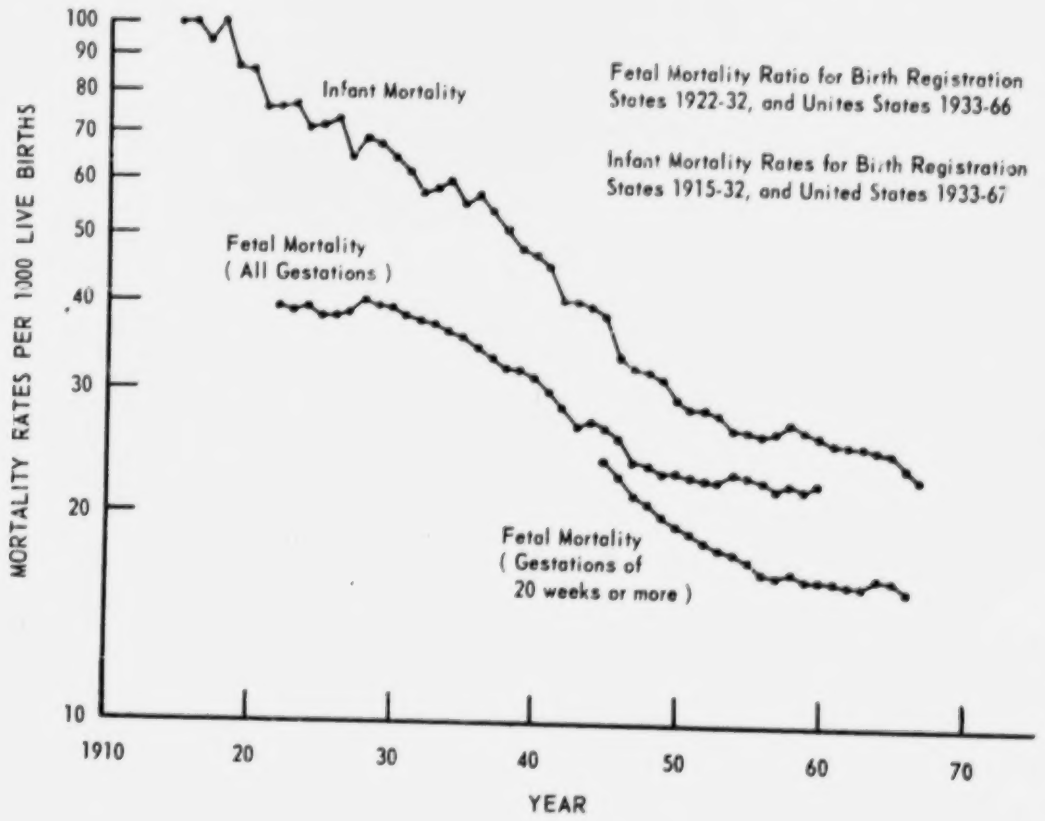


Figure 6. Infant mortality rates 1915-1967 and fetal mortality ratios 1922-1966, for the United States.

The analysis of fetal mortality ratios presents additional problems due to changes in definition of fetal death, differences in State legal requirements for reporting fetal deaths, and changes in coding of fetal deaths during the years Dr. Sternglass is using. As of 1965, there were seven different periods of gestation at which fetal death registration was required in the various vital statistics registration areas in the United States; all periods of gestation, after 16 weeks, advanced to 20th week, 20 weeks or more, after 20 weeks, advanced to the 5th month, and after 5th month. Before 1945, all fetal deaths reported were included in the "all gestations" rates published for the United States. During the period 1945-1960 rates for gestations of 20 weeks or more as well as for all gestations were reported; since 1960, only data on fetal deaths at gestations of 20 weeks or more have been published. Since 1945, all reported fetal deaths with an entry of "unknown" gestation time have been included in the group "20 weeks or more". Although this is probably a valid assumption for those areas requiring registration of fetal deaths only at 20 weeks or more gestation, it is probably not valid for areas requiring registration of all fetal deaths, regardless of period of gestation.

For these reasons, any use of fetal mortality ratio data to compare trends within States, between States, or in the United States as a whole must be made with extreme caution. Variations in the requirements for fetal death registration among States, variation in registration completeness in relation to reporting requirements, and coding problems in relation to the period of gestation for certain States all contribute to the problems of making comparisons among States and over time. It should be noted that these qualifications regarding fetal mortality data are stated in each publication of such data by the National Center for Health Statistics.

FETAL MORTALITY

Accepting the limitations on the use of fetal mortality ratios for comparisons of trends, there are some other comments which should be made about the use of fetal mortality data presented by Dr. Sternglass as evidence of a genetic effect due to the incorporation of ^{90}Sr in the genetic material of the parents.

Figure 7 (from Sternglass) is a semi-logarithmic plot of reported fetal mortality ratios for the three counties of the Albany-Troy-Schenectady, N. Y. area for the years 1937-1966.

Shown on this plot are lines which are stated to "indicate trend of average, minimum, and maximum rates." Although an average rate can be calculated, it is difficult to interpret a trend of the single points which represent the minimum and maximum rates observed, and yet it it these lines in Figure 7 which appear to support the thesis of a change occurring within a year or two following the rainout in 1953.

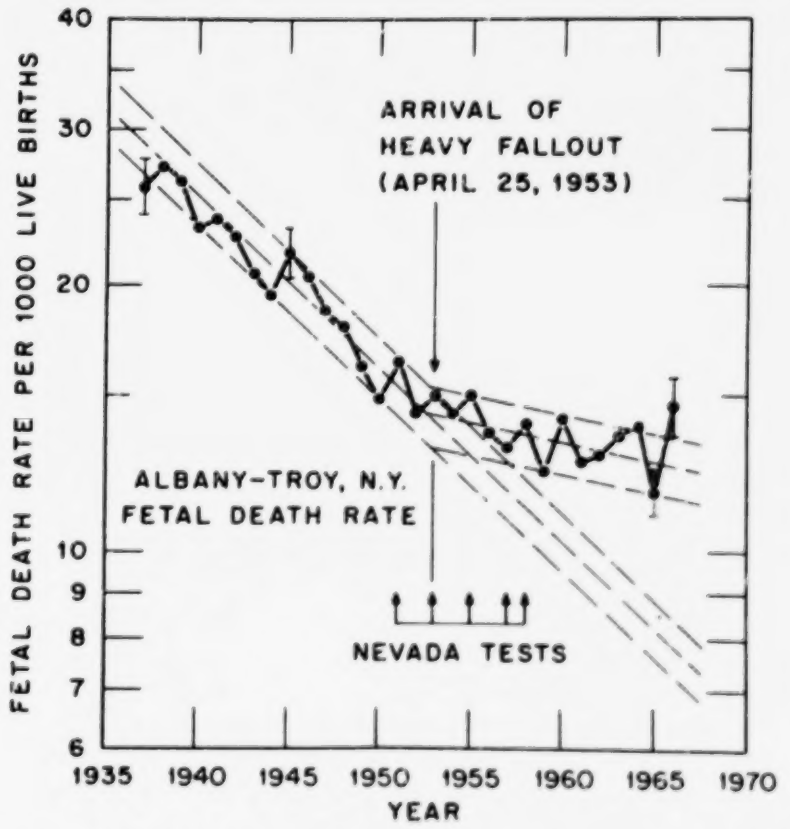


Figure 7. Fetal death rate reported per 1000 live births vs. time in the Albany-Troy, N.Y., area before and after the arrival of fallout 36 hrs after the detonation of a 43-kt weapon on Apr. 25, 1953. Dashed lines indicate trend of average, minimum and maximum rates. (from Sternglass)

The data points included in Figure 7 have been replotted and a least-squares line fitted to the data for the years 1937-1952 (Figure 8). Also, parallel lines bisecting the maximum and minimum points observed in these years have been drawn. This plot appears to indicate that the change in slope of decline of fetal mortality in the Albany-Troy area started as early as 1952 and was not established until 1958.

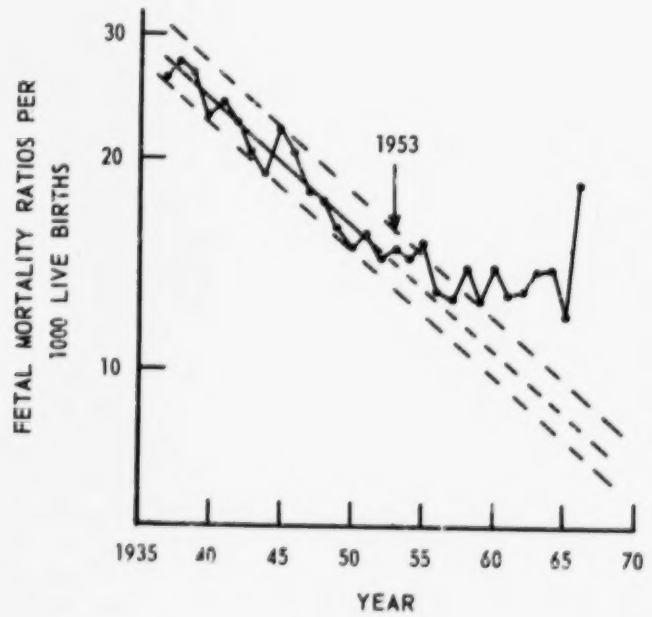


Figure 8. Fetal mortality ratios for the Albany-Troy area as read from Figure 7, with a line fitted to the values for the years 1937 - 1952 and parallel lines entered which bisect the ratios during these years with maximum deviations from the line.

Dr. Sternglass extends his investigation of fetal mortality rates by attempting to "see whether changes in fetal death rate appeared not only in the Troy-Albany area but also in New York State as a whole, and whether subsequent tests are also reflected in changes of the fetal mortality." In Figure 9 (from Sternglass) he has graphed the fetal mortality ratios for New York State and for California which, he states, "has a similar population pattern but received less fallout because of its low rainfall and its location west of the Nevada Test Site, away from the direction of the prevailing winds." He states that the rates in New York State "began to deviate from the 1935-1950 rate of decline between 1950 and 1952, the period in which atmospheric weapons tests began at the Nevada Test Site" while "California maintained its steady decline" until "2 to 3 years after the onset of the hydrogen bomb tests in the Pacific in 1954."

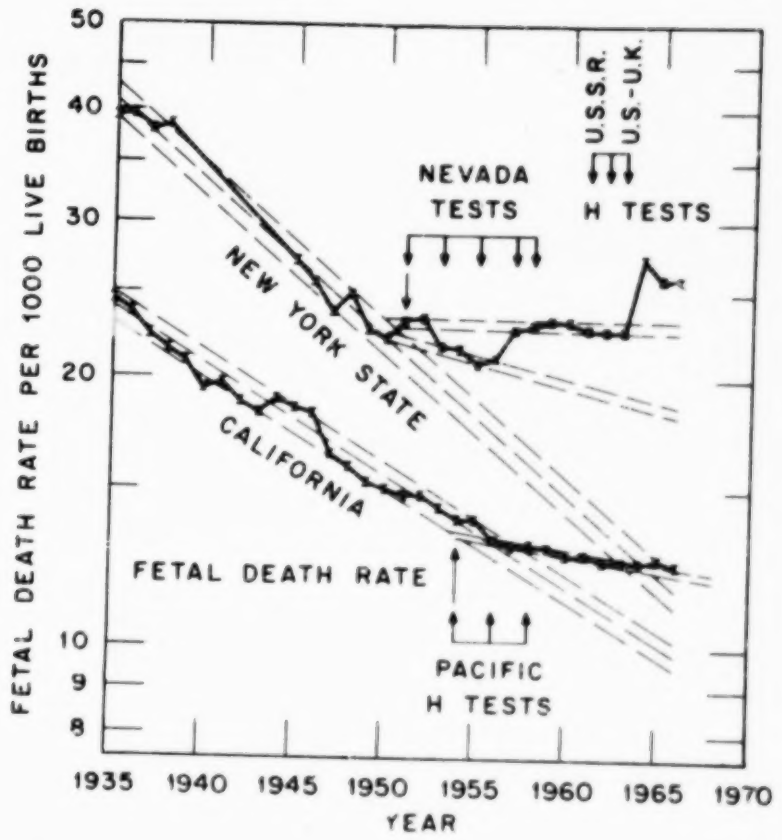


Figure 9. Fetal deathrate reported per 1000 live births vs. time for New York State and California before and after the onset of nuclear weapons tests in Nevada in 1951. Symbols indicate the standard deviation due to statistical fluctuations alone. (from Sternglass)

The interpretation of this plot must be questioned on several counts. New York State vital statistics come from two separate Health Departments, that of New York State exclusive of New York City, and of New York City. These two jurisdictions have different requirements for fetal death registration, and the variability in completeness of reporting between these two departments over the years in question makes any interpretation of fetal mortality ratios for New York State as a whole extremely difficult. Although the data for the years 1939-1944 have been omitted from Figure 9, the erratic character of the fetal mortality ratios for New York State are clearly shown when all of the reported data are plotted (Figure 10). A least-squares line has been fitted to the data points for 1935-1950, but any attempt to describe the trend of these reported rates with a single line is highly suspect, and to define a time of change of trend from this line is virtually impossible. The California data relate only to a single registration area and, hence, do not present the same problem of interpretation as those from New York State. The fetal mortality ratios for California have been plotted and a line fitted to the data for the years 1935-1950 (Figure 10). It appears that the trend in California probably started to shift in the early 1950's rather than in 1956-1957 as suggested by Dr. Sternglass.

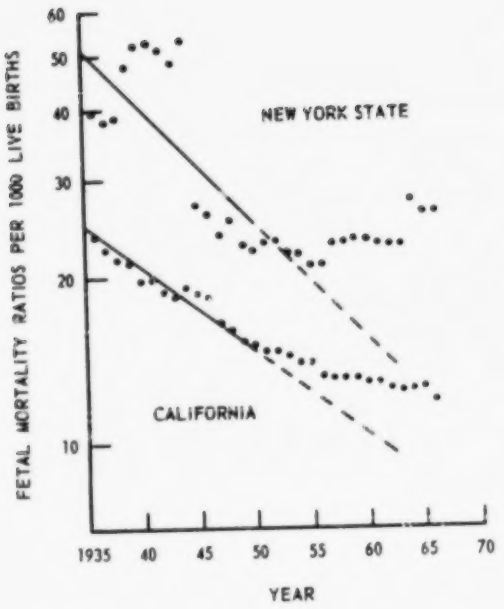


Figure 10. Fetal mortality ratios for New York State and California for the years 1935 - 1967 with lines fitted to the ratios for the years 1935 - 1950.

Finally, Dr. Sternglass attempts to show a correlation between the cumulative deposition of ^{90}Sr in New York State and the excess of fetal deaths per 1000 live births, obtained by subtracting the observed rate in New York as plotted in Figure 9 from the extrapolated rate of his arbitrary line (Figure 11 - from Sternglass). Given the problems inherent in the data from which these excess fetal death rates were derived, their use to establish a correlation of this type is questionable.

Dr. Sternglass concludes that there is an association between fetal mortality and deposition of fallout, and continues his argument by attempting to demonstrate that the isotope primarily responsible is ^{90}Sr. The strontium data he uses are those which were measured in the mandibular bone of third trimester fetuses aborted in the St. Louis, Mo., area as reported by Rosenthal, et al. 1/

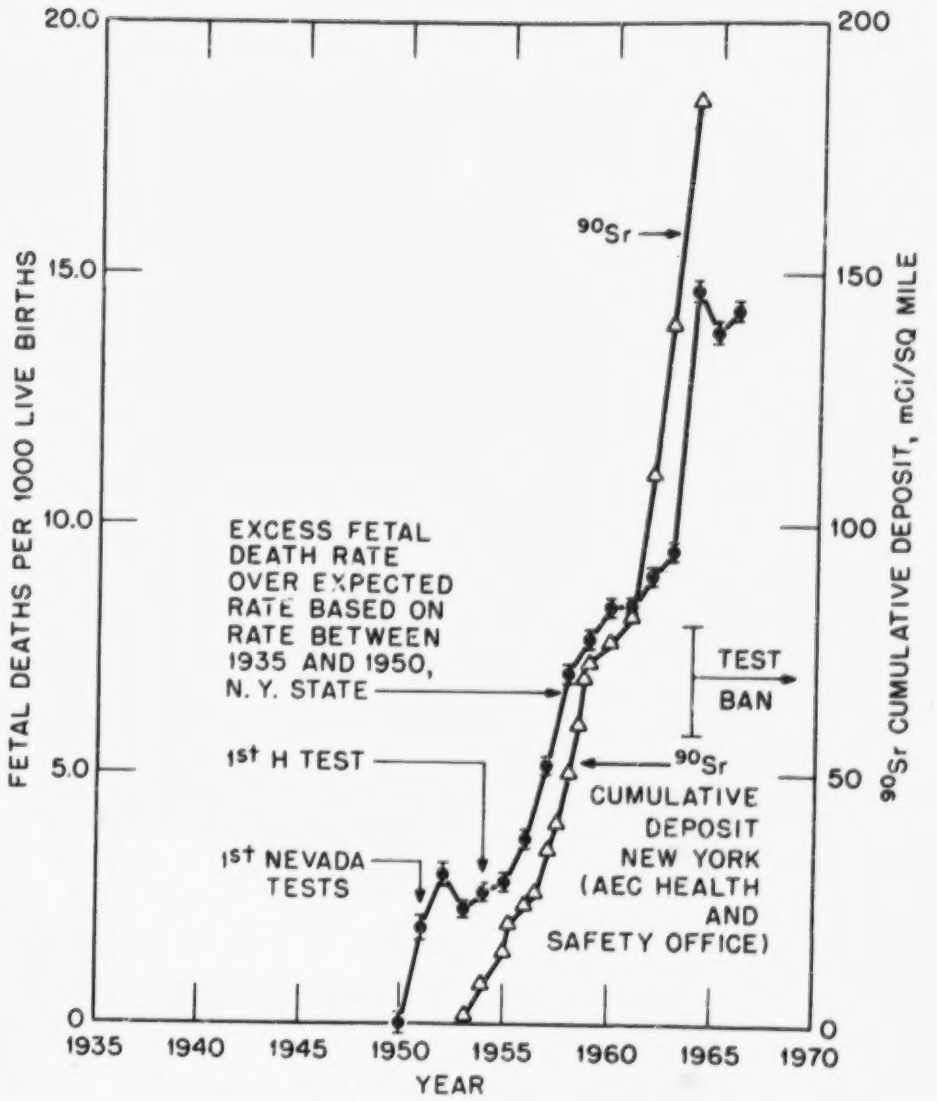


Figure 11. Excess fetal death rate in New York State over the rates expected from the 1935 - 1950 rate of decline, as shown in Figure 9, and the cumulative deposition of ^{90}Sr on the ground in the New York area vs. time, in years, before and after the start of atmospheric weapons tests in Nevada. (from Sternglass)

There is some confusion between his text and his figures as to the derivation of "excess" fetal mortality. The fetal mortality ratios, 1935-1966, for the State of Missouri with a line fitted to the 1935-1950 observations are plotted and the "excess" deaths over the projection of the 1935-1950 trend line corrected to the year 1959 are entered, as well as the time of atomic test series (Figure 12 - from Sternglass). The text states that the fetal mortality excess in the St. Louis, Mo., area was obtained from this figure and has been plotted against the ^{90}Sr in fetal mandibular bone as reported by Rosenthal (Figure 13 - from Sternglass). However, the legend of Figure 12 correctly states that the "excess" is for the State of Missouri.

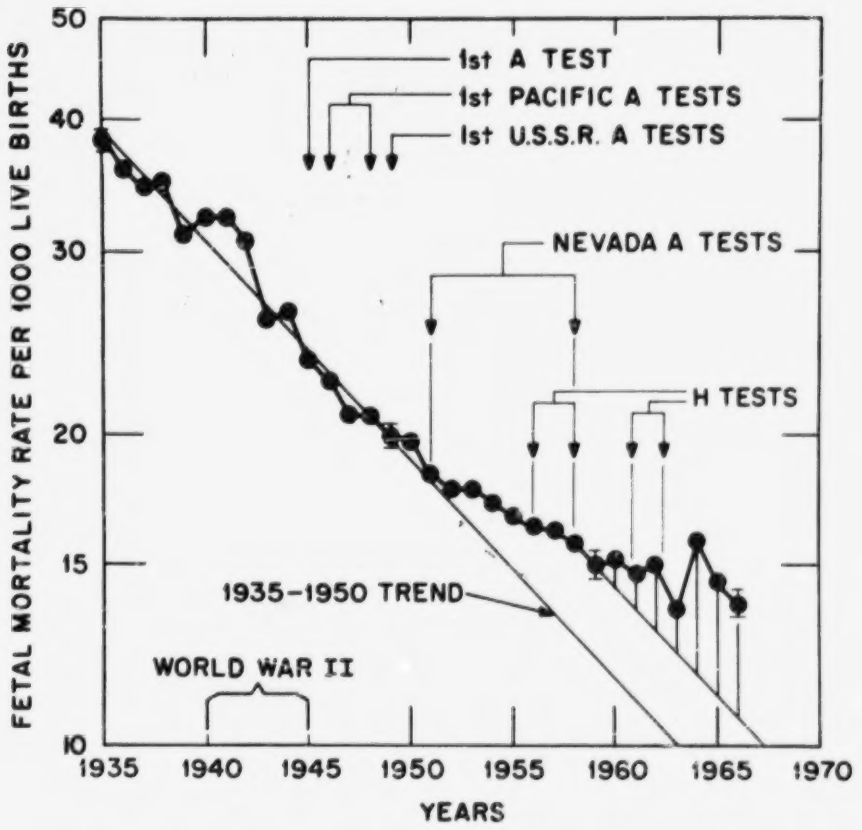


Figure 12. Fetal death rate for Missouri showing the excess over the expected rate based on the 1935 - 1950 regression line. Data from U. S. Vital Statistics. (from Sternglass)

It should also be noted that there are two observations on ^{90}Sr in fetal bone for each year 1962-1965 with one in the last half of 1961 and one in the first half of 1966. Dr. Sternglass does not explain why he connects only some of the points when drawing the line showing the pattern of the ^{90}Sr data. (Dr. Rosenthal used all points in his publication from which these data were drawn.) There is also an apparent error in the "excess" fetal deaths as entered for the year 1963. We believe the correct value should be 1.2 rather than the 2.2 plotted. The data have been re-plotted connecting all the ^{90}Sr points and making the correction in the point for the 1963 excess fetal deaths (Figure 14). Dr. Sternglass points out a strong correlation between the rise and fall of the ^{90}Sr in fetal mandibular bone and the "excess" fetal deaths. However, he also goes on to state that there is "an indication of about a year's lag in the rise of fetal mortality relative to the rise in the ^{90}Sr bone concentration." Although this year lag appears for 1962, 1963, and 1964, the pattern is lost in 1965 and 1966 when the pattern of "excess" fetal mortality parallels that of ^{90}Sr concentration.

Since the data on "excess" fetal mortality are based on the entire State of Missouri rather than only the St. Louis area, the interpretation of this demonstrated correlation is valid only if the fetal mortality ratios for the State of Missouri and the St. Louis area are identical and therefore interchangeable. This is not the case. The ratios for 1935-1966 for the State of Missouri and those for 1942-1966 (the only years for which data are available) for the city and county of St. Louis have been plotted on a semi-logarithmic scale (Figure 15). If the hypothesized association between excess fetal death and ^{90}Sr in fetal bone is valid, the correlation using St. Louis data should show even a closer relationship than that using fetal death data for Missouri. The "excess" fetal deaths for the period 1960-1966 for St. Louis City and County have been derived using the same method employed by Dr. Sternglass and again plotted against the ^{90}Sr in fetal mandibular bone (Figure 16). Instead of showing a closer relationship, the fetal death excess now appears to rise and fall independently of the ^{90}Sr in bone.

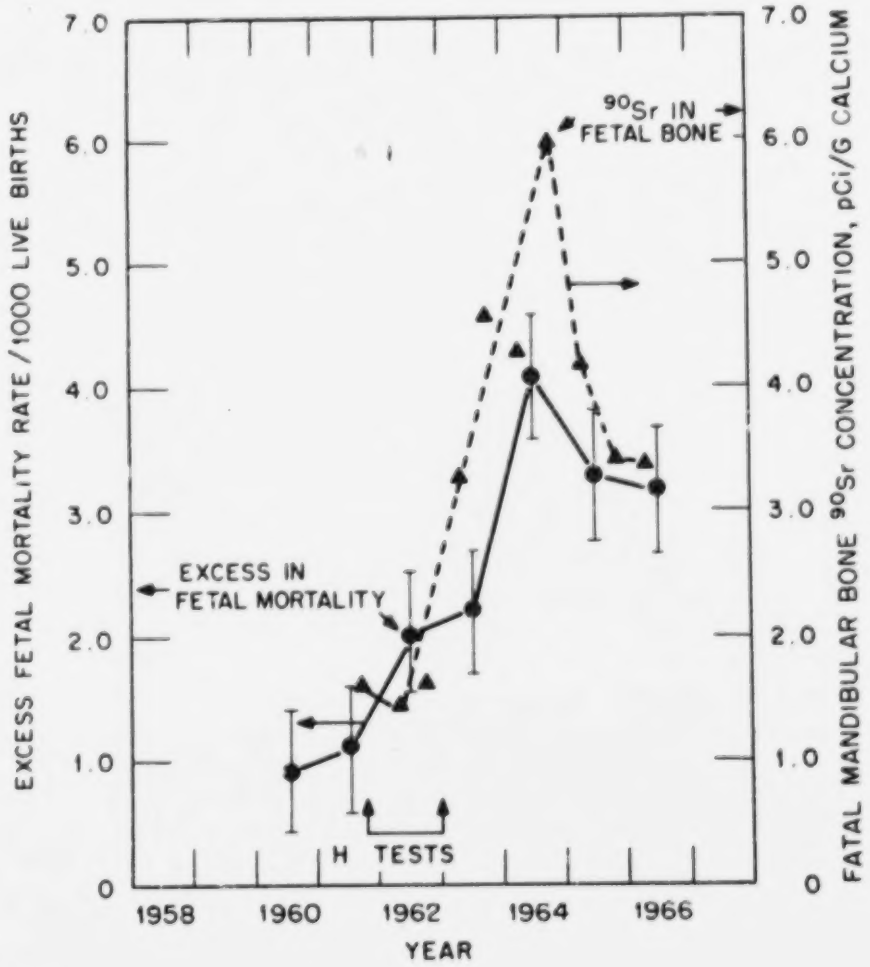


Figure 13. Excess fetal mortality in Missouri relative to the 1959 value declining at the 1935 - 1950 rate compared with the ^{90}Sr content of fetal mandibular bone measured in the St. Louis, Mo., area by Rosenthal et al. for 62 aborted fetuses. (from Sternglass)

Dr. Sternglass concludes that he has demonstrated an association between ^{90}Sr deposition and the change in rate of decline of fetal death ratios, and that the most probable explanation of the mechanism responsible is the incorporation of the ^{90}Sr into the genetic material as a result of its particular chemical valency, resulting in more fetal deaths. For the reasons we have suggested, the validity of the stated association appears questionable.

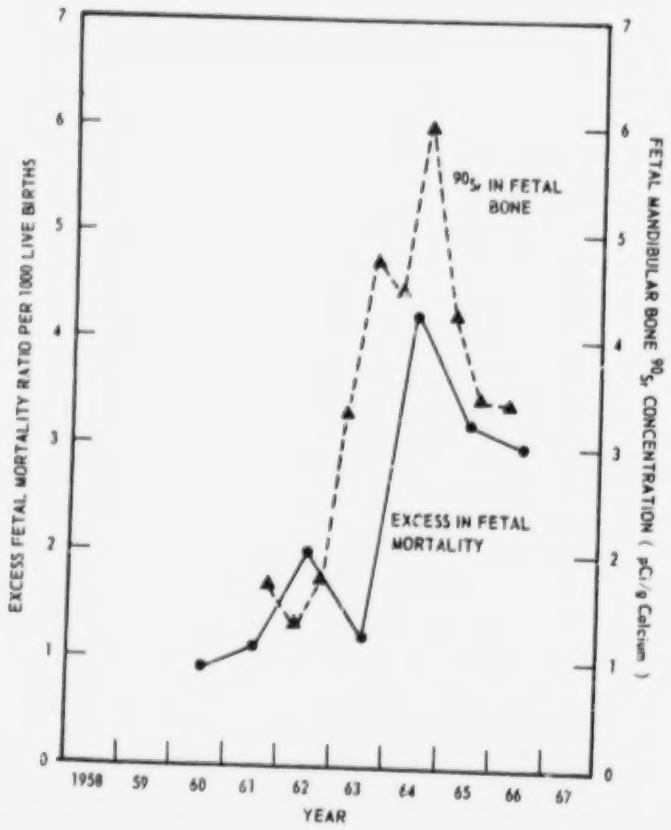


Figure 14. Excess fetal mortality in Missouri relative to the 1959 value declining at the 1935 - 1950 rate compared with the ^{90}Sr content of fetal mandibular bone measured in the St. Louis, Mo., area by Rosenthal, et al. for 62 aborted fetuses.

Figure 15. Fetal mortality ratios for the State of Missouri and the city and county of St. Louis, Mo., for the years 1935 - 1966.

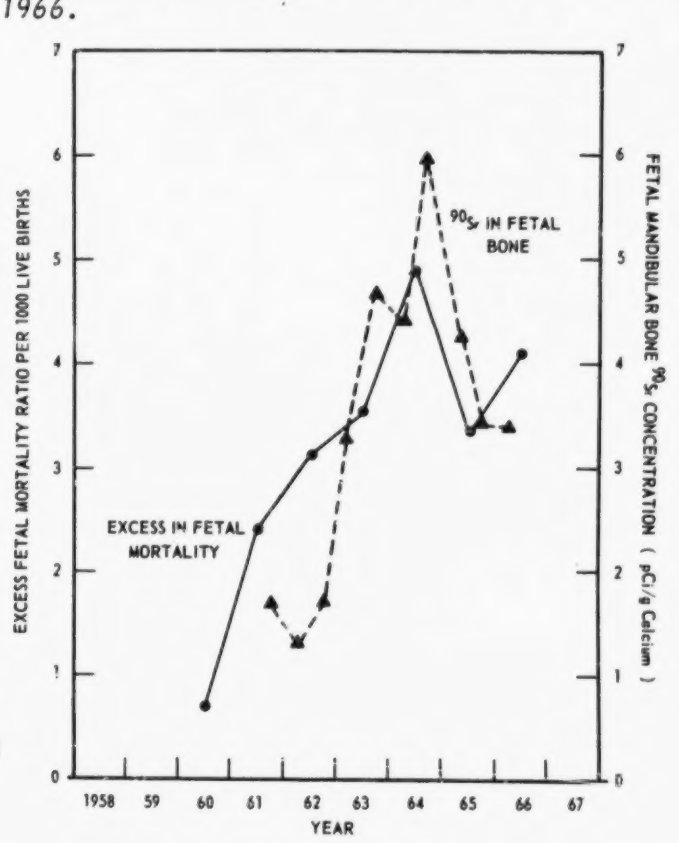


Figure 16. Excess fetal mortality in St. Louis City and County relative to the 1959 value declining at the 1942 - 1950 rate compared with the ^{90}Sr content of fetal mandibular bone measured in the St. Louis, Mo., area by Rosenthal, et al. for 62 aborted fetuses.

INFANT MORTALITY

The infant mortality data for the various States as well as for the United States do not present the same problems in interpretation as do the fetal mortality data. Dr. Sternglass begins his investigation of the possible relationship of changes in rates of decline of infant mortality and ^{90}Sr with a comparison of the "percent excess" of infant mortality in the State of Missouri for the years 1948-1958 relative to the 1935-1950 rate of decline and the ^{90}Sr concentration in deciduous second molars of children in the St. Louis area as reported by Rosenthal, et al. (Figure 17 - from Sternglass). He explains that "the curves are closely parallel, and the excess mortality rises somewhat earlier than the ^{90}Sr during the first few years of the tests in Nevada when the short-lived ^{89}Sr was proportionally more important." Although the trend line in Figure 17 shows a steady rise in the percent excess of infant mortality, the actual data points show a dip in 1951. It appears that Dr. Sternglass has also utilized a faulty arithmetic procedure when calculating his percent excesses relative to 1948. The percent excess for each year is derived by subtracting the expected cases, as predicted by the fitted line, from the observed cases and dividing the excess cases by the expected cases. In order to determine the percent excess relative to the 1948 value set to zero, he has simply subtracted the calculated percent for 1948 from the calculated percent for each subsequent year and entered this on his graph. As each of these percents is based on a different denominator, such a subtraction does not yield a meaningful result.

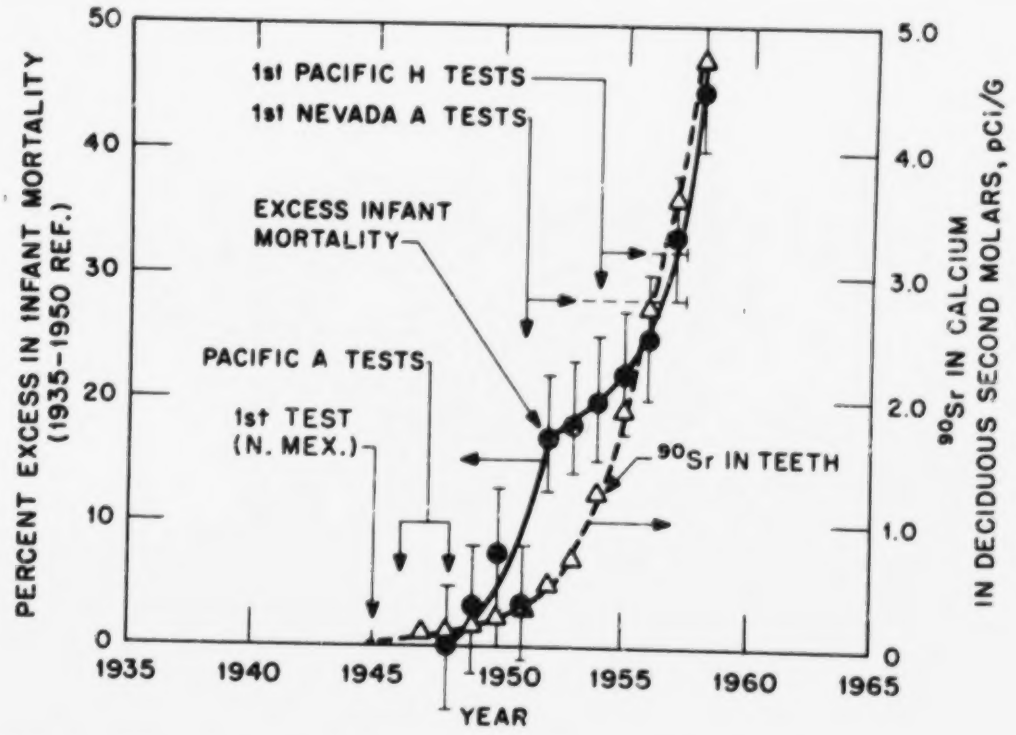


Figure 17. Excess infant mortality (0 to 1 year) in Missouri relative to the 1935 - 1950 rate of decline compared with the change in ^{90}Sr concentration in deciduous second molars for children in the St. Louis area as reported by Rosenthal et al. for the period 1948 to 1958. (from Sternglass)

An attempt was made to reevaluate this correlation correcting the arithmetic error. However, it was observed that, as with fetal mortality, the infant mortality rates for Missouri and for St. Louis City and County are not identical. Since infant mortality rates for the St. Louis area are available only for the years beginning with 1942, a line was fitted to the 1942-1950 data for St. Louis and another fitted to the Missouri data for the same time period and the percent excesses calculated relative to the 1948 value in each case. If infant mortality is predictable based on trends of past years, the change in the years included to fit the projected line should not appreciably change the projection, and the St. Louis City and County data should fit the ^{90}Sr data more closely than the Missouri data if the hypothesis of an association is valid. The recalculated percent excesses for Missouri and St. Louis City and County and the ^{90}Sr concentration in deciduous second molars for children in the St. Louis area as reported by Rosenthal, et al.[2/] have been plotted (Figure 18). It is evident that the correction of the arithmetic error and the use of the 1942-1950 data for the projected line has made a considerable change in the Missouri data. The St. Louis data are difficult to explain under the Sternglass hypothesis. There is certainly nothing in the ^{90}Sr data to explain the sharp rise in percent excess for the years 1950-1952 nor the leveling-off in 1954-1956.

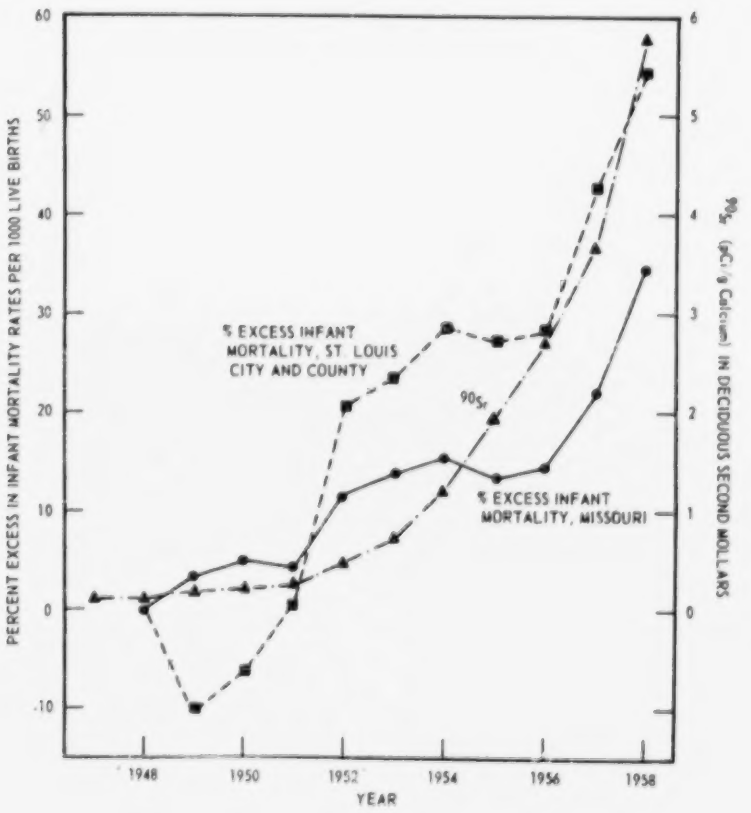


Figure 18. Percent excess infant mortality in Missouri and St. Louis City and County relative to the 1942 - 1950 rate of decline compared with the change in ^{90}Sr concentration in deciduous second molars for children in the St. Louis area as reported by Rosenthal, et al. for the period 1948 to 1958.

Dr. Sternglass next states that "the onset of Nevada tests in 1951 also appears to be reflected in the infant mortality rates for the United States as a whole" (Figure 19 - from Sternglass). He states that "a strong leveling trend in the rate of decline for the total population occurred after 1951 and did not end until 2 years after the cessation of atmospheric testing in 1963, when a decline at about the rate for the 1935-1950 period resumed." Again, there appear to be some differences between the points shown in Figure 19 and the published data on infant mortality for the United States, and the trend line appears not to be a fitted line.

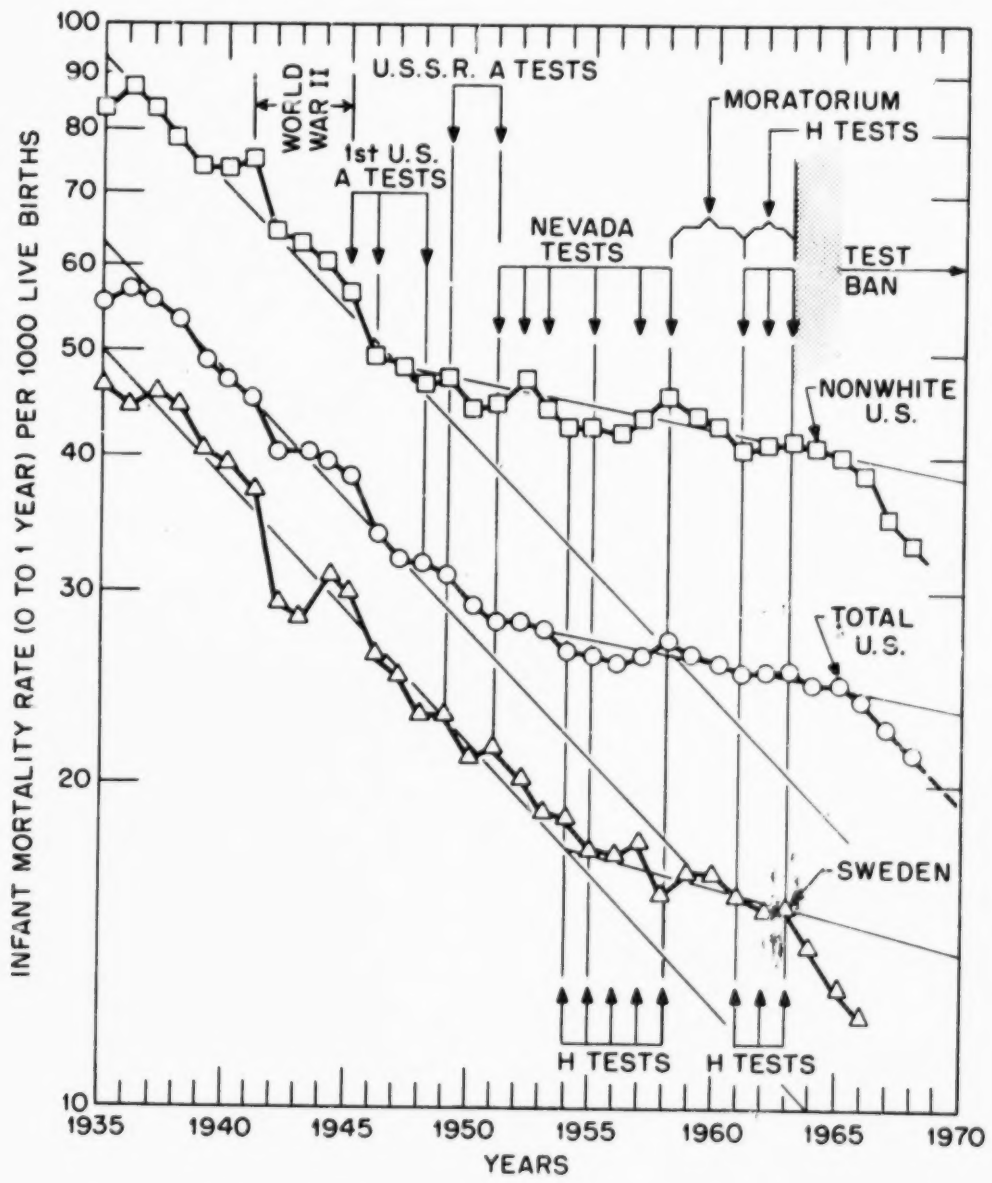


Figure 19. Infant mortality rates per thousand live births for the United States and Sweden for the period 1935 - 1968. Rates are shown both for the total population of the United States and the nonwhite population of the United States separately. The 1967 and 1968 "nonwhite U. S." points and the 1965 and 1966 "Sweden" points were added in proof. (from Sternglass)

The same data for the total United States population have been replotted using the correct points and with the trend lines for 1935-1950 and 1951-1965 fitted by the least-squares technique. A line parallel to the 1935-1950 trend line has been entered for the years 1966-1967 (Figure 20). This figure does show the change in trend in 1951 as pointed out by Dr. Sternglass. He then points out that the nonwhite population shows a similar leveling which started earlier, "within 2 to 3 years after the early New Mexico and Pacific tests of 1945-1948, whose fallout reached predominately the southern regions of the United States with their high concentration of nonwhite population and high annual rainfall." The only evidence for this trend appears to be a line drawn parallel to the one which was arbitrarily selected for the total United States population. The data for United States nonwhite infant mortality are also plotted in Figure 20 with a line fitted to the years 1935-1950, and it fails to show the earlier leveling trend in this sub-population.

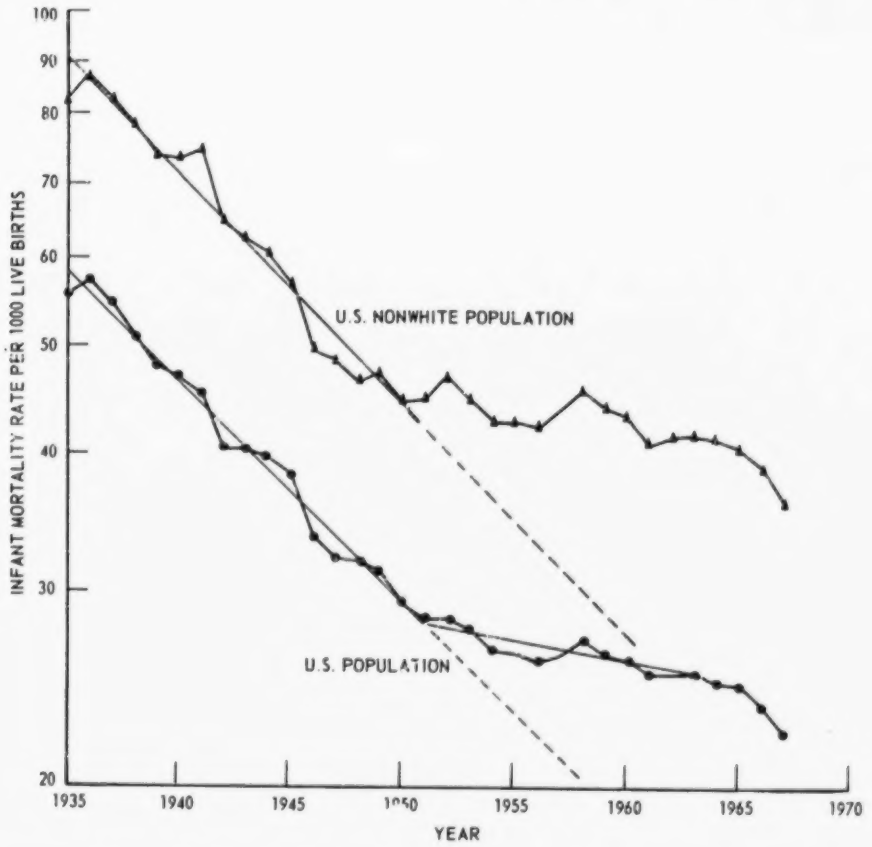


Figure 20. Infant mortality rates for the United States and for the nonwhite population of the United States for the years 1935 - 1967. Lines have been fitted to the rates for the years 1935 - 1950 and 1951 - 1965 for the total United States population. A line has been fitted to the rates for the years 1935 - 1950 for the nonwhite population.

It could be argued that if a significant part of the nonwhite population of the United States had been affected by the mid-40's weapons testing, the trend line should be fitted only to the points before the first test in 1945 in order to investigate a possible change. To appropriately compare the experience of the nonwhite population with that of the white population, the infant mortality data for the United States for the white population and the nonwhite population separately, with trend lines fitted to the appropriate data for 1935-1945, have been plotted and the times of various atomic test series noted (Figure 21). It is interesting that trend lines based on the years before fallout existed seem to suggest that there was no real change in trend in the white population until 1954 or 1955, a full 9 to 10 years after testing began. Contrary to the expectation based on results of early United States testing as hypothesized by Dr. Sternglass, the observed infant mortality rates for nonwhites are all lower than those expected, and continue to be so until after 1951.

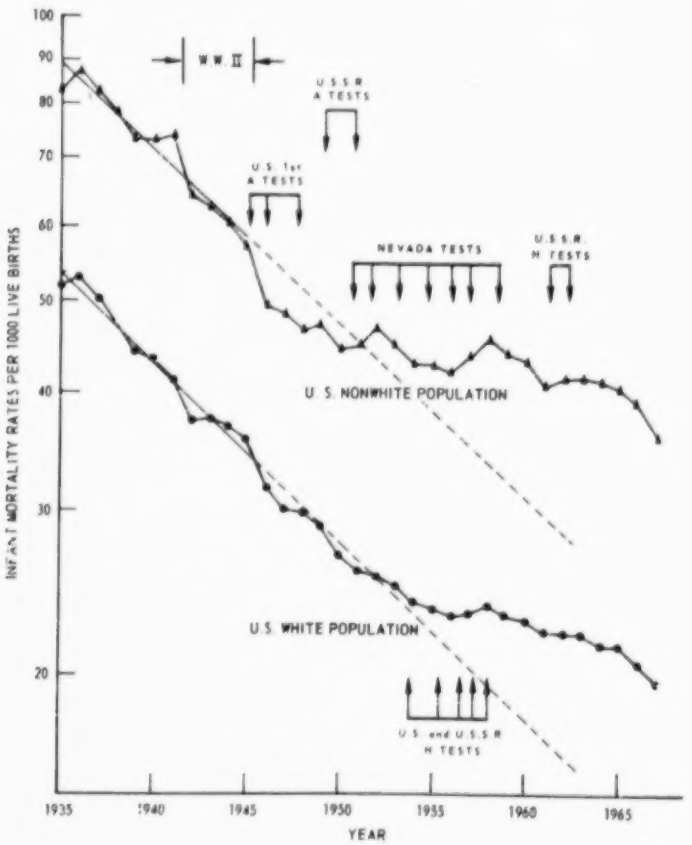


Figure 21. Infant mortality rates for the United States white population and the United States nonwhite population for the years 1935 - 1967 with lines fitted to the rates for the years 1935 - 1945.

Dr. Sternglass continues his discussion of Figure 19 by pointing out "that there appears to exist a period of 3 to 5 years during which the trends change following either the onset or cessation of testing, suggesting a buildup and healing or elimination of damage to the reproductive cells." A careful study of the data in his plot (Figure 19) or in that for the white and nonwhite populations (Figure 21) does not show any such consistent pattern either within a population group or between groups. This is particularly evident in the trend lines for the periods of moratorium and test ban in 1959-1961 and 1963-1967 with the high yield USSR test series inbetween.

The last point Dr. Sternglass makes regarding Figure 19 is to show Sweden's infant mortality rates in support of the thesis that there is no "natural plateau" in the attainable infant mortality rate. However, he goes on to state that Sweden, like other European countries, has shown a leveling trend "beginning with the onset of large hydrogen weapons testing in the late 1950's." Again, the line drawn on Sweden's rates is simply parallel to the one selected for the United States. The same Swedish infant mortality rates with the addition of the years 1965-1966 and with a trend line fitted to the 1935-1950 data have been plotted (Figure 22).

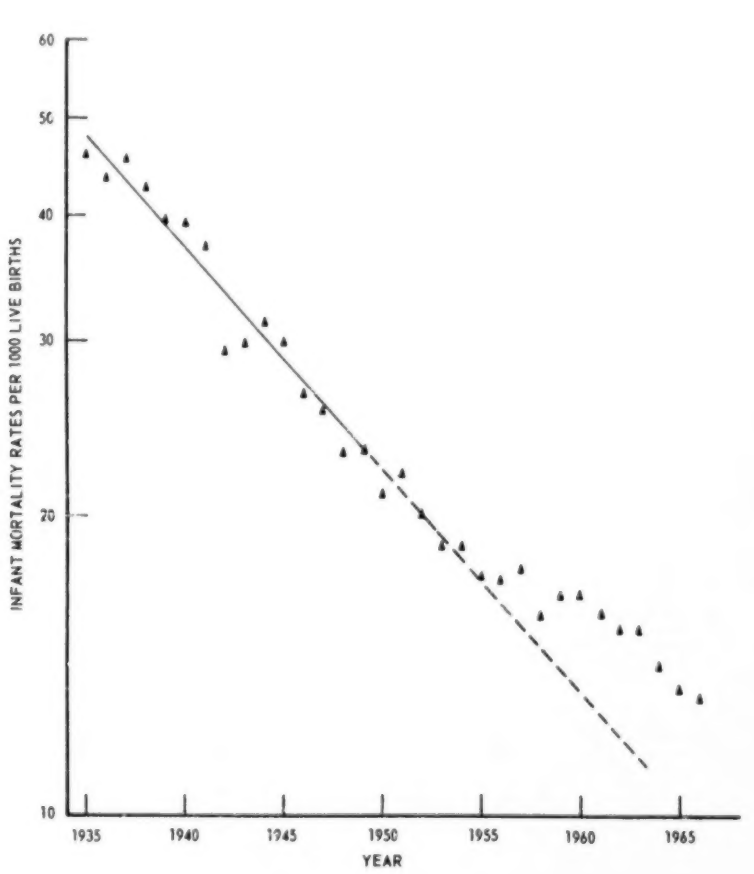


Figure 22. Infant mortality rates for Sweden for the years 1935 - 1966 with a line fitted to the rates for the years 1935 - 1950.

With the fitted line and the additional years of data, it is more difficult to accept Dr. Sternglass' interpretation of the data. The first point which seriously deviates from the projected line is 1957. This would imply a 3-year delay in observable change after the onset of large weapons testing in the Pacific in 1954. However, the 1958 point is close to the projected line, and by 1961 the infant mortality rate again seems to be dropping at a rate similar to that projected. If the 4 years of change in rate of decline in 1957-1960 in the Swedish infant mortality rates are indeed a reflection of deposition of ^{90}Sr from the large tests in the Pacific, there is no explanation as to why the large series of Russian tests in the fall of 1961 and 1962, which led to the world-wide deposition of more ^{90}Sr than any other series, is not reflected in the infant mortality in Sweden in 1965 and 1966.

Figure 23 from Sternglass shows "the degree of correlation of the leveling of infant mortality for the United States and the increase of ^{90}Sr produced by large weapons testing." Even if one accepts the hypothesis of a relationship between the deposition of ^{90}Sr in the United States and the leveling trend in infant mortality, this does not necessarily lead to an assumption that infant mortality is related to the total strontium produced, independent of where it was deposited. The same comment must be made regarding the correlation between "excess" infant mortality in the United States and the deposition of ^{90}Sr in the entire northern hemisphere of the world.

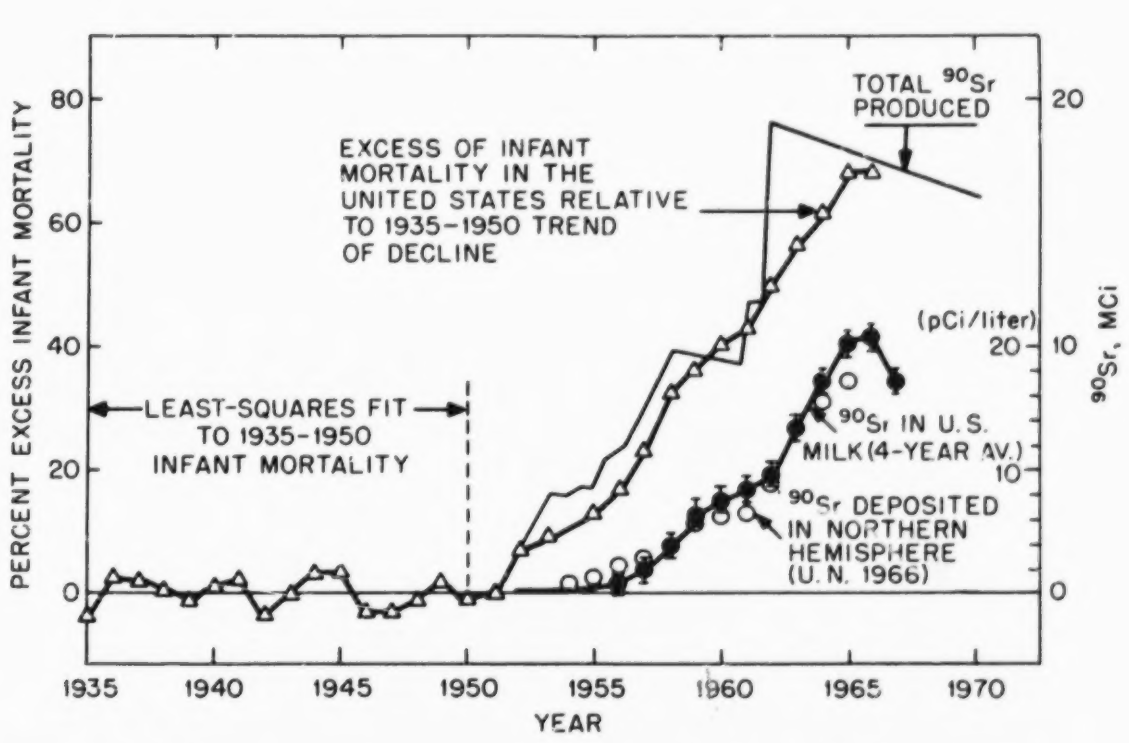


Figure 23. Percent excess infant mortality relative to the 1935 - 1950 regression line for the period 1950 to 1966. Also plotted is the total amount of ^{90}Sr produced, the ^{90}Sr deposited in the northern hemisphere, and the 4-year ^{90}Sr average content in U. S. milk. (from Sternglass)

A further analysis by Dr. Sternglass relates to measurements of ^{90}Sr in milk. The Public Health Service Pasteurized Milk Network has 62 stations located in the 50 states, the District of Columbia, and Puerto Rico. The samples for measurement are taken at the market place in the cities comprising the network. The ^{90}Sr content of the milk is, of course, dependent on the deposition of ^{90}Sr on pastures, as well as on the feeding practices used in the particular milk shed areas supplying milk to the cities. There is tremendous variability, therefore, in the ^{90}Sr in milk from city to city. For example, in 1964, the year of peak readings, the annual averages for the various stations ranged from 4 to 54 pCi per liter. Thus, any attempt to average values from all of the stations to arrive at a "U.S. milk content" of ^{90}Sr without weighting the values according to the amount of milk produced in the area of each station is of doubtful utility.

The "3- to 5-year delay in the peak of infant mortality, which suggests a genetic rather than a direct somatic effect," which Dr. Sternglass finds, is according to him, best shown in the changes in rates of mortality in the southern "downwind" states following the first atomic weapons test in Alamogordo, N. Mex., in July 1945 (Figure 24 - from Sternglass). We have further tested, using his method of calculation, Dr. Sternglass' hypothesis that these eight southern States were, in 1950, reflecting changes in rates of infant mortality resulting from fallout. If his hypothesis is correct, one would expect to see no "excess" in the infant mortality rates in these States until 1948, followed by a rapid buildup in 1949 and 1950. The "percent excess infant mortality," relative to the rate of decline for 1940-1945, was calculated for the 48 continental States and the District of Columbia for each of the years 1946-1951. In 1947, Arkansas and Louisiana already showed an "excess" infant mortality of 5% or greater, but Montana did not. In 1948, Texas, Mississippi, and Alabama joined the 5% or greater group, but, so did Wyoming and South Dakota. In 1949, Georgia, South Carolina, and North Carolina passed the 5% level, as did Virginia and Vermont, while South Dakota now showed less than 5% "excess". (According to our calculations, Montana only had a 4.4% "excess" rather than the 7% shown.) In 1951, Virginia was again over the 5% level. Thus, only the year 1950 shows the distribution of States with "percent of excess mortality" of 5% or greater which Dr. Sternglass uses as a demonstration of a 3- to 5-year delay from the arrival of fallout to a 5% "excess infant mortality". No explanation other than radiation from fallout is suggested for these "excesses" including even the 1946 "excesses" in Montana and North Dakota. In order for the Sternglass hypothesis of a 3- to 5-year delay effect to stand unchallenged some source of radioactive strontium in Montana and North Dakota must be identified in the years 1941-1943 (the Hanford, Washington, reactor did not go into operation until December 1944); the first atomic weapons test in 1945 could not explain the changes in Arkansas and Louisiana as early as 1947, and still other sources would be needed to explain the changes in Vermont, South Dakota, Wyoming, and Virginia. We are unaware of any such sources.

Figure 24. Geographical distribution of excess infant mortality in the United States relative to the values expected on the basis of the 1940 - 1945 rate of decline. (a) For 1946, 1 year after the test in New Mexico. (b) For 1950, 5 years after the test. Note the absence of any evidence for a direct radiation effect in utero or early infancy in the 1946 plot, suggesting that the major effect is genetic in nature. Also note the consistent excess in the north central states known to have had high ^{90}Sr levels accumulated by 1950 - 1958. (from Sternglass)

As there does not appear to be any reasonable hypothesis to explain the discrepancies already noted in the years between 1946 and 1950, it is probably not necessary to pursue further the question of validity of the relationship between infant mortality in the South and the first atomic weapons test. However, the use of the years 1940-1945 for the basic trend line rather than the years 1935-1945 may be questioned. (All of the other calculations were based on lines fitted to data starting in 1935.) An effort was made to try to understand this shift in base years by repeating the calculations for the 48 States and the District of Columbia using trend lines fitted to the data for 1935-1945. The results have been entered on maps similar to the maps presented by Dr. Sternglass (Figure 25). As is apparent, the use of the additional years of data suggest a somewhat different pattern than that in Figure 24.

Plots showing "the detailed correlation with ^{90}Sr in the milk of various areas of the United States differing in rainfall and geographic location relative to the test sites" and "percent excess infant mortality" is the next evidence for an association which Dr. Sternglass presents (Figure 26 - from Sternglass). The Public Health Service Raw Milk Network was established in 1957-1958 with ten stations, the Pasteurized Milk Network with its 62 stations in 1961-1962. Nine stations were in both networks. Comparison studies were published by the Public Health Service between the ^{90}Sr readings in the two networks, and it was shown that ^{90}Sr values from the Raw Milk Network could be used with those of the Pasteurized Milk Network for all of the stations except Atlanta, Ga., and St. Louis, Mo. Although Dr. Sternglass has included these two States among the six he has selected for study, no explanation regarding the source of ^{90}Sr data for the early years is given. He has also repeated the error of subtracting percents based on different denominators and reporting these values as "percent excess infant mortality". The data were all recalculated correctly for the four States with valid readings of ^{90}Sr in milk and plotted, including all of the 1967 infant mortality points. The same procedure was followed and the data plotted for the other three States in the network which Dr. Sternglass did not analyze, namely, Ohio, California, and Washington (Figure 27). Only the data for two of the seven States plotted, Illinois and Texas (and possibly Utah), would appear to support the hypothesis of a direct relationship between the 4 - year average values of ^{90}Sr in milk and "percent excess infant mortality". It is very difficult to try to explain why the State of Washington has the highest ^{90}Sr values and the lowest "excess" infant mortality. Conversely, California has the lowest ^{90}Sr values and yet has one of the highest "excess" infant mortality curves. The data for New York and Ohio indicate instances of rising ^{90}Sr values, while the related infant mortality curves are declining.

The last evidence for an association between ^{90}Sr deposition and "percent excess infant mortality" presented by Dr. Sternglass is an attempt to show that a scattergram of 4-year running averages of ^{90}Sr in United States milk (corrected for 1/10 the activity of ^{89}Sr in United States milk) vs. percent excess infant mortality in the United

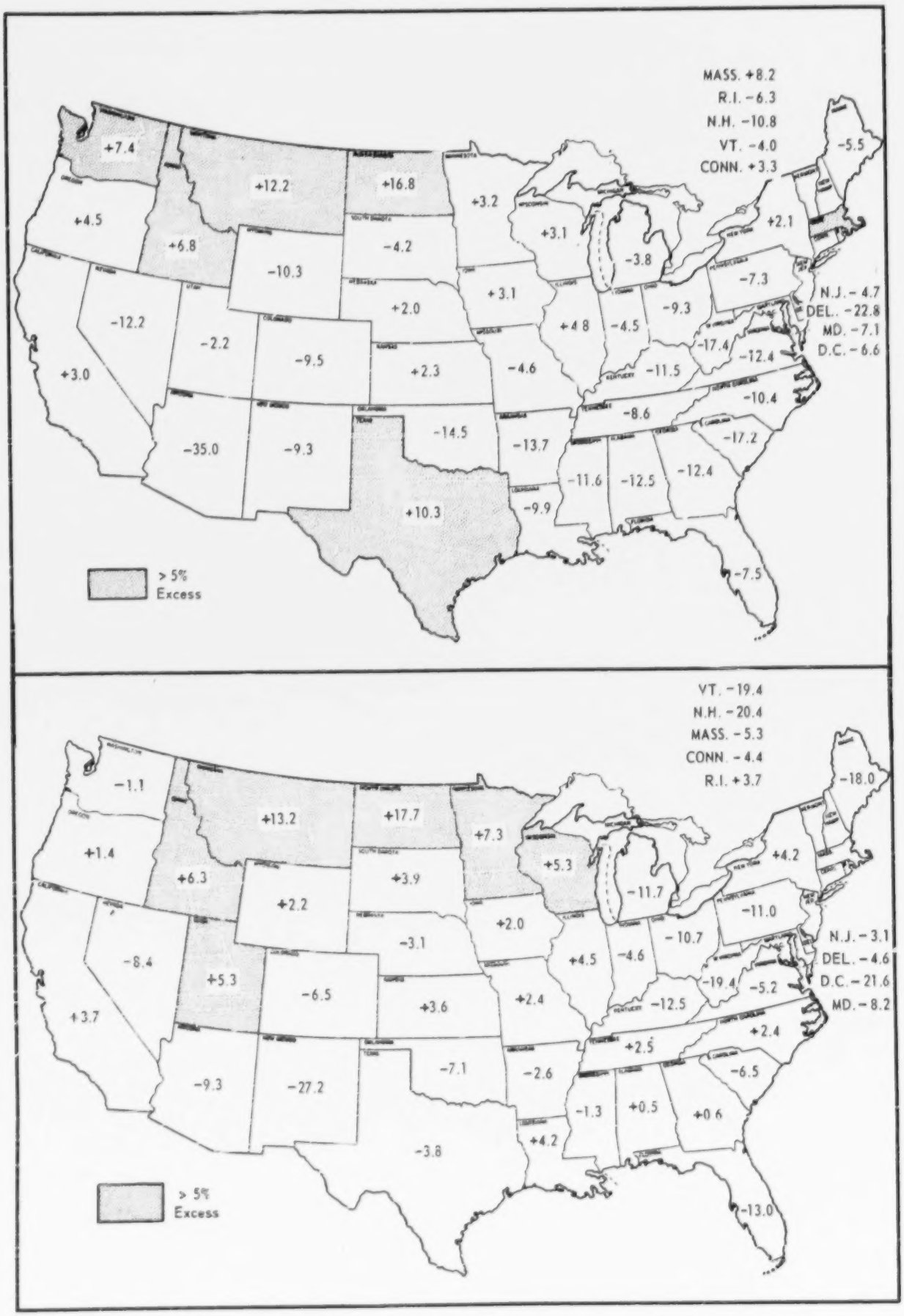


Figure 25. Geographic distribution of percent excess infant mortality relative to the 1935 - 1945 rate of decline. (Top) 1946, (Bottom) 1950.

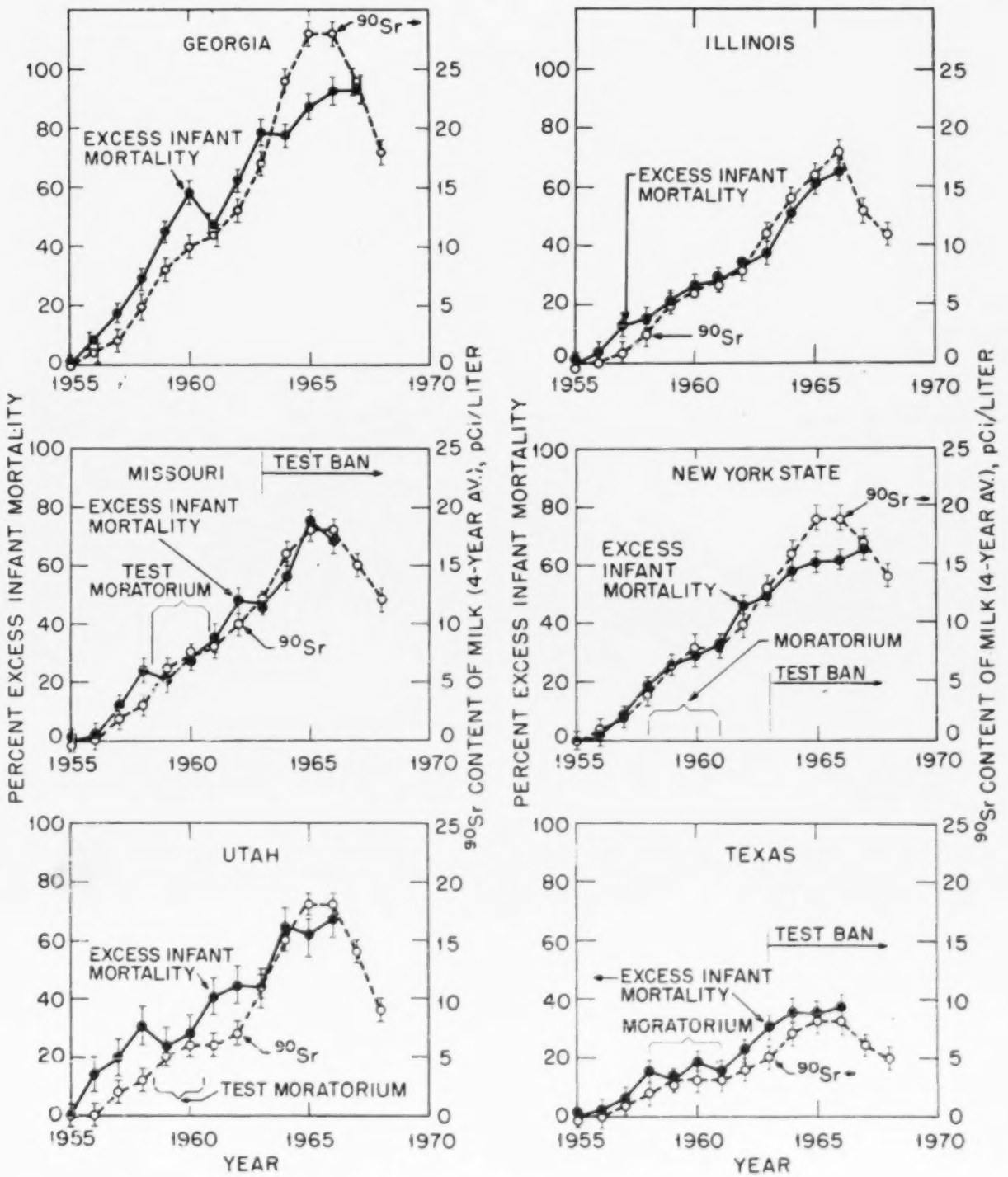


Figure 26. Percent excess infant mortality (0 to 1 year) and 4-year average ^{90}Sr content of milk for Georgia, Missouri, Utah, Illinois, New York, and Texas. The excess mortality rate has been calculated relative to the 1955 value, declining at the 1935 - 1950 rate of decrease before major weapons testing. (from Sternglass)

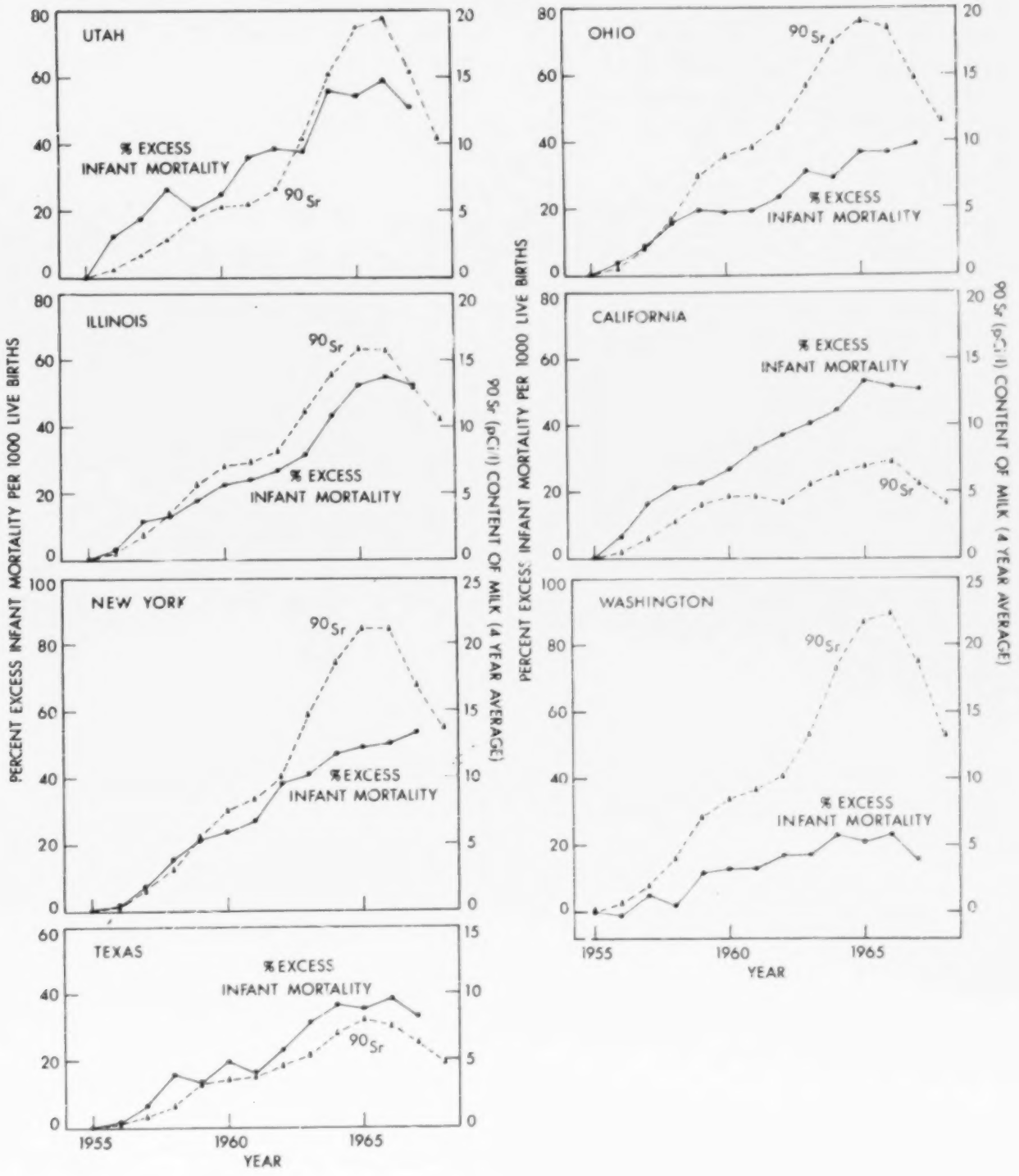


Figure 27. Percent excess infant mortality relative to the 1955 value declining at the 1935-1950 rate and the 4-year average ^{90}Sr concentrations in milk in Utah, Illinois, New York, Texas, Ohio, California and Washington.

States is linear (Figure 28 - from Sternglass). The difficulty in using any single number for ^{90}Sr in all United States milk has been discussed earlier. The same problem exists for the ^{89}Sr measurements. Once again, Dr. Sternglass has miscalculated the percent excess infant mortality. Given the definite lack of association between percent "excess" infant mortality and ^{90}Sr in milk demonstrated on a State-by-State basis earlier, further discussion of this plot appears fruitless.

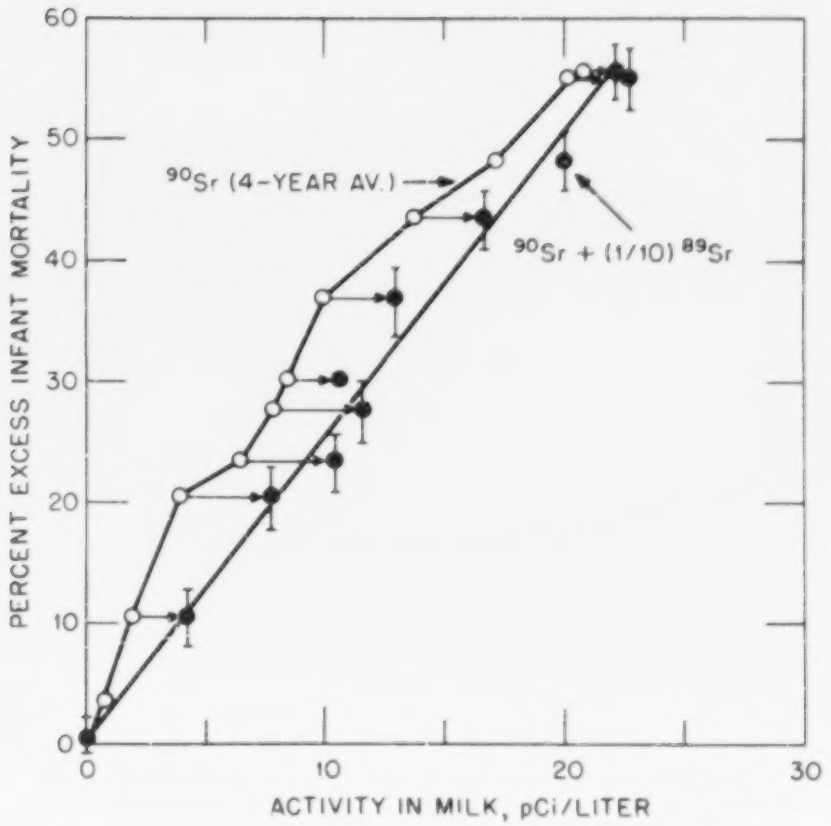


Figure 28. Excess infant mortality in the United States relative to the 1955 value declining at the 1935 - 1950 rate of decline vs. the ^{90}Sr activity in the milk, with and without the added contribution due to (1/10) of the ^{89}Sr activity. The activities, in picocuries per liter, are 4-year moving averages based on the Public Health Service Pasteurized Milk Network Measurements reported in Radiation Health Data and Reports. (from Sternglass)

DISCUSSION

Although all the evidence which Dr. Sternglass has presented to support an association between ^{90}Sr deposition and a decrease in the rate of decline of infant and fetal mortality in the United States has failed to stand up under careful scrutiny, the important implications of such an association, if true, warrant some further investigation. This investigation has been limited to infant mortality data due to the problems inherent in the fetal mortality data.

If the changes in infant mortality rates are primarily the result of a single environmental contaminant, that is, fallout from atomic weapons testing affecting the genetic material of the parents, then within a State, one would expect the time of change in trend to coincide for both neonatal and postneonatal deaths. One could also hypothesize that, in those States with a significant proportion of nonwhite births, the change in trend in the infant mortality rates for the white and nonwhite populations should also coincide in time. However, recognizing that most of the ^{90}Sr enters the human body via the food chain, principally in milk, it is possible that the generally lower socioeconomic levels of the nonwhite population could be associated with a lower milk consumption than the white population, and therefore, the nonwhite population might be expected to show a change in trend later than that of the white population in the same State.

Before this hypothesis could be tested, an objective means to determine the point of inflection in trend had to be developed. With the assumption that two straight lines fitted to the logarithmic transformations of the rates adequately describe the data for the period 1935-1967 (an admitted oversimplification) it was possible to fit two lines to all the data points starting with 1935-1939 and 1940-1967 and, continuing in a systematic manner, adding the data for 1 year to the first line and at the same time dropping it from the second line, to compute all pairs of lines through the set of points representing 1935-1962 and 1963-1967. The time of change was determined by selecting that pair of lines which had the maximum difference in slope and the minimum sum of squares around both lines. The results of these calculations are shown in Table 1. The States with no entry either had no change or there was more than one change in trend. It can be seen that, in many States, the neonatal and postneonatal rates did change in the same year, but in some there was a difference in the time of change, and in some cases by a significant number of years.

The results of the comparison of the time of change of the white and nonwhite populations in the 26 States with more than 10% nonwhite births in 1960 are even more striking. Contrary to expectation, the time of change of the nonwhites preceded the whites in 14 States; in one State the difference was as much as 5 years. Thus, the hypothesis that the time of change of trend for the white and nonwhite populations coincide must be rejected.

Table 1. Year of Maximum Change of Infant Mortality Rates

State	Neonatal Mortality Rates	Post Neonatal Mortality Rates	Infant Mortality Rates White	Infant Mortality Rates Nonwhite
Alabama	1947	1949	1952	1948
Arizona	- -	- -	1947	- -
Arkansas	1948	1948	1951	1948
California	- -	1950	1950	1950
Colorado	- -	1951	1950	
Connecticut	1950	1950	1951	
Delaware	- -	1951	1951	1950
District of Columbia	1949	1950	- -	1950
Florida	1951	1950	1951	1950
Georgia	1949	1949	1950	1949
Idaho	1953	1951	1951	
Illinois	1951	1951	1951	1950
Indiana	1950	1953	1951	
Iowa	1952	1950	1952	
Kansas	1949	1950	1951	
Kentucky	- -	1955	- -	
Louisiana	1950	1950	1950	1950
Maine	1952	1952	1952	
Maryland	1951	1950	1951	1951
Massachusetts	1951	1950	1950	
Michigan	1949	1953	1949	1951
Minnesota	1951	1951	1951	
Mississippi	1950	1948	1951	1948
Missouri	1950	1950	1950	1950
Montana	1953	1950	1950	
Nebraska	- -	1950	1950	
Nevada	- -	- -	- -	- -
New Hampshire	1951	1951	1951	
New Jersey	1951	1950	1950	1950
New Mexico	- -	- -	- -	- -
New York	1950	1951	1951	1949
North Carolina	1950	1948	1952	1948
North Dakota	1945	1949	1950	
Ohio	1951	1951	1950	1951
Oklahoma	- -	1950	1951	1946
Oregon	- -	1950	1950	
Pennsylvania	1950	1952	1951	1951
Rhode Island	1949	1950	1950	
South Carolina	1953	1949	1952	1949
South Dakota	1950	1950	1950	
Tennessee	1952	1952	1952	1951
Texas	1952	1953	1953	1950
Utah	1950	1949	1950	
Vermont	1950	1951	1951	
Virginia	1950	1950	1951	1948
Washington	- -	1950	- -	
West Virginia	1954	1953	1953	
Wisconsin	1952	1950	1950	
Wyoming	- -	1951	- -	

The states with no entry had no change or there was more than one time of change.

Another possible hypothesis is that the accumulated "excess" deaths per thousand, as calculated by Dr. Sternglass, should correlate with the accumulated ^{90}Sr deposition on a State-by-State basis, if there is a relationship between infant mortality and ^{90}Sr. There are 13 States for which complete strontium deposition data are available. The correlation coefficient of the accumulated "excess" deaths, as calculated by Dr. Sternglass, and the total ^{90}Sr deposited is -0.015. Thus, no evidence of a relationship is found.

The rate of decline of infant mortality in the United States did change around 1950. This lowering of the rate of decline has been a concern of many people working in the public health field. Many careful studies have been made, and no single factor nor group of factors have been identified which explain this change. While the hypothesis that this change is a result of ^{90}Sr deposition from fallout is an interesting one, the data presented do not appear to indicate any relationship between the change in rate of decline of infant mortality and the deposition of fallout in the United States.

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